=> fil hcaplu
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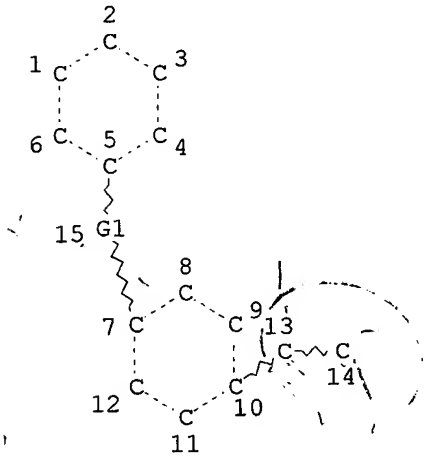
FILE COVERS 1907 - 8 Aug 2002 VOL 137 ISS 6
FILE LAST UPDATED: 7 Aug 2002 (20020807/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

CAS roles have been modified effective December 16, 2001. Please check your SDI profiles to see if they need to be revised. For information on CAS roles, enter HELP ROLES at an arrow prompt or use the CAS Roles thesaurus (/RL field) in this file.

=> d stat que
L1 STR




VAR G1=O/S/N
NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:

RSPEC I
NUMBER OF NODES IS 15

STEREO ATTRIBUTES: NONE

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L3        41052 SEA FILE=REGISTRY SSS FUL L1
L4        44349 SEA FILE=HCAPLUS L3
L5          640 SEA FILE=HCAPLUS L4 (L)((BLOOD OR BLD)(5A)(GLUCOSE OR SUGAR OR
                TRIGLYCERIDE? OR PRESSURE?) OR ?DIABET? OR ?TRIGLYCER? OR
                ?HYPERTEN?)
L6         1079 SEA FILE=HCAPLUS L4 (L)(?MEDIC? OR ?PHARM? OR ?THERAP? OR
                ?DRUG?)
L7           43 SEA FILE=HCAPLUS L6 AND L5
L8       455228 SEA FILE=HCAPLUS THU/RL
L9           56 SEA FILE=HCAPLUS L5 AND L8
L11          90 SEA FILE=HCAPLUS L9 OR L7
L12          69 SEA FILE=HCAPLUS L11 NOT 2002/PY
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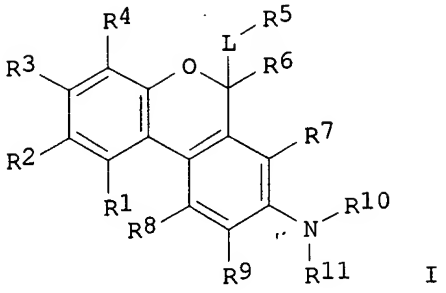
=> d ibib abs hitrn l12 1-69

L12 ANSWER 1 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:833866 HCAPLUS
DOCUMENT NUMBER: 135:371633
TITLE: Preparation of 6H-dibenzo[b,d]pyran derivatives as glucocorticoid receptor antagonists for treatment of diabetes
INVENTOR(S): Kym, Philip R.; Lane, Benjamin C.; Pratt, John K.; Geldern, Tom Von; Winn, Martin; Brenneman, Jehrod; Patel, Jyoti R.; Arendsen, David L.; Akritopoulou-zanze, Irini; Ashworth, Kimba L.; Hartandi, Kresna
PATENT ASSIGNEE(S): Kym, Philip, USA
SOURCE: U.S. Pat. Appl. Publ., 94 pp., CCont.-in-part of U.S. Ser. No. 654,322.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| US 2001041802 | A1 | 20011115 | US 2001-795998 | 20010228 |
| US 6329534 | B1 | 20011211 | US 2000-654322 | 20000901 |

PRIORITY APPLN. INFO.: US 1999-151839P P 19990901
US 2000-654322 A2 20000901
US 1999-388251 A1 19990901
OTHER SOURCE(S): MARPAT 135:371633
GI



I

AB The title compds. [I; R1 = alkanoyl, CN, halo, etc.; R2 = H, R1; R3, R4, R7-R9 = H, R1; L = a bond, alkylene; R5 = alkanoyl, alkoxy, aryl, etc.; R6 = H, alkyl; LR5 and R6 together = A(CH2)d (wherein d = 1-4; A = CH2, O, S, etc.) to form a spiro ring; R10, R11 = H, alkyl, aryl, etc.], useful for treating type II diabetes, obesity, hyperglycemia, inadequate glucose clearance, hyperinsulinemia, hypertriglyceridemia, and high-circulating glucocorticoid levels, were prepd. E.g., a multi-step synthesis of I [R1 = OMe; R2-R4 = H; L = a bond; R5 = 3-F3CC6H4; R6 = H; R7 = Me; R8, R9 = H; R10 = SO2Me; R11 = H] which showed 82.1% GR binding inhibition at 1.7 .mu.M, was given.

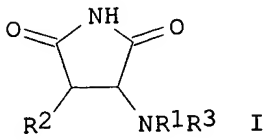
IT **373622-75-4P**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. of 6H-dibenzo[b,d]pyran derivs. as glucocorticoid receptor antagonists for treatment of **diabetes**)

L12 ANSWER 2 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:747748 HCAPLUS
DOCUMENT NUMBER: 135:288688
TITLE: Pyrrole-2,5-dione derivatives for the treatment of diabetes
INVENTOR(S): Haigh, David; Slingsby, Brian Peter; Smith, David Glynn; Ward, Robert William
PATENT ASSIGNEE(S): Smithkline Beecham P.L.C., UK
SOURCE: PCT Int. Appl., 67 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2001074771 | A1 | 20011011 | WO 2001-EP3687 | 20010402 |

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ,

VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.: GB 2000-8264 A 20000404
OTHER SOURCE(S): MARPAT 135:288688
GI



I

AB The title compds. I [R1 = substituted or unsubstituted carbocyclic or heterocyclic arom. ring, which ring may be fused to a substituted or unsubstituted carbocyclic or heterocyclic arom. or non-arom. ring; R2 = substituted or unsubstituted carbocyclic or heterocyclic arom. ring, which ring may be fused to a substituted or unsubstituted carbocyclic or heterocyclic arom. ring, with the proviso that R2 is not 3-indolyl or a fused-ring deriv. of 3-indolyl; R3 = H, or R1 and R3 together with the nitrogen atom to which they are attached form a fused substituted or unsubstituted heterocyclic ring], inhibitors of GSK-3, were prepd. E.g., a mixt. of 3-(4-aminophenylthio)phenylacetic acid, 3-chloro-4-(4-chlorophenyl)-1H-pyrrole-2,5-dione, and 1-methyl-2-pyrrolidinone was heated in a sealed tube in a hotblock set at 690C for 28.5 h to give 3-[4-[3-(carboxymethyl)phenylthio]phenylamino]-4-(2,3-difluorophenyl)-1H-pyrrole-2,5-dione.

IT **365244-98-0P 365245-00-7P 365245-09-6P**
**365245-43-8P 365245-69-8P 365245-90-5P**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(pyrrole-2,5-dione derivs. for the treatment of **diabetes**)

REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:689256 HCAPLUS
DOCUMENT NUMBER: 136:48684
TITLE: Triiodothyronine concomitantly inhibits calcium overload and postischemic myocardial stunning in diabetic rats
AUTHOR(S): Oshiro, Yoshito; Shimabukuro, Michio; Takasu, Nobuyuki; Asahi, Tomohiro; Komiya, Ichiro; Yoshida, Hisashi
CORPORATE SOURCE: Second Department of Internal Medicine, Faculty of Medicine, University of the Ryukyus, Okinawa, 903-0215, Japan
SOURCE: Life Sciences (2001), 69(16), 1907-1918
CODEN: LIFSAK; ISSN: 0024-3205

PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Acute effects of triiodothyronine (T3) on postischemic myocardial stunning and intracellular Ca2+ contents were studied in the isolated working hearts of streptozotocin-induced diabetic rats and age-matched controls. After two weeks of diabetes, serum T3 and T4 levels were decreased to 62.5% and 33.9% of control values. Basal preischemic cardiac performance did not differ between diabetic and control rats. In contrast, during reperfusion after 20-min ischemia, diabetic rats exhibited an impaired recovery of heart rate (at 30-min reperfusion 57.5% of baseline vs. control 88.5%), left ventricular (LV) systolic pressure (44.1% vs. 89.5%), and cardiac work (23.1% vs. 66.0%). When 1 and 100 nM T3 was added before ischemia, heart rate was recovered to 77.2% and 81.8% of baseline, LV systolic pressure to 68.3% and 81.9%, and cardiac work to 50.8% and 59.0%, resp. Diabetic rat hearts showed a higher Ca2+ content in the basal state and a further increase after reperfusion (4.96.+-.1.17 vs. control 3.78.+-.0.48 .mu.mol/g, p<0.01). In diabetic hearts, H+ release was decreased after reperfusion (5.24.+-.2.21 vs. 8.70.+-.1.41 mmol/min/g, p<0.05). T3 administration caused a decrease in the postischemic Ca2+ accumulation (1 nM T3 4.66.+-.0.41 and 100 nM T3 3.58.+-.0.36) and recovered the H+ release (1 nM T3 16.2.+-.3.9 and 100 nM T3 11.6.+-.0.9). T3 did not alter myocardial O2 consumption. Results suggest that diabetic rat hearts are vulnerable to postischemic stunning, and T3 protects the myocardial stunning possibly via inhibiting Ca2+ overload.
IT **51-48-9**, T4, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(triiodothyronine concomitantly inhibits calcium overload and postischemic myocardial stunning in **diabetic** rats)
IT **6893-02-3**, Triiodothyronine
RL: PAC (Pharmacological activity); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(triiodothyronine concomitantly inhibits calcium overload and postischemic myocardial stunning in **diabetic** rats)
REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 4 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:564981 HCAPLUS
DOCUMENT NUMBER: 135:152623
TITLE: Synthesis of aryl-alkenyl-oxy-arylpropionic acid derivs. and their use in treatment of PPAR mediated disorders including diabetes and obesity
INVENTOR(S): Mogensen, John Patrick; Sauerberg, Per; Bury, Paul Stanley; Jeppesen, Lone; Pettersson, Ingrid
PATENT ASSIGNEE(S): Novo Nordisk A/S, Den.
SOURCE: PCT Int. Appl., 224 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|

| | | | | |
|---|---|---|---|---|
| WO 2001055085 | A1 | 20010802 | WO 2001-DK58 | 20010126 |

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: DK 2000-136 A 20000128
DK 2000-1071 A 20000707
DK 2000-1594 A 20001025

OTHER SOURCE(S): MARPAT 135:152623

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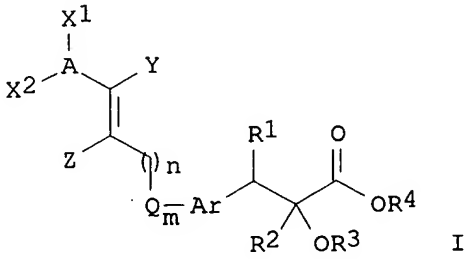


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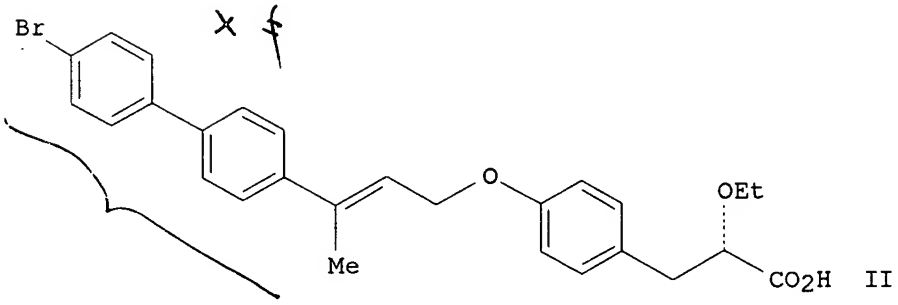


II

AB Title compds. I [A = (un)substituted (hetero)aryl; X1-2 = H, (un)substituted (hetero)aryl; Y = H, alk(en/yn/enyn)yl, (hetero)aralkyl; Z = H, halo, OH, alkyl, etc.; Q = O, S, N-; Ar = (hetero)arylene or a divalent heterocyclic group; R1 = H, OH, halo or forms a bond with R2; R2 = H, alkyl or forms a bond with R1; R3 = H, alk(en/yn/enyn)yl, aryl, aralkyl, etc.; R4 = H, alk(en/yn/enyn)yl, aryl; n = 0 - 3; m = 0 - 1] were prepd. Over 150 synthetic examples were disclosed. For instance,

4-(4-bromophenyl)acetophenone was reacted with triethylphosphonoacetate to give E-3-(4'-bromobiphen-4-yl)but-2-enoic acid Et ester in 80% yield. The enoate was converted to the corresponding allylic alc. (DIBAL-H, PhMe) and used to alkylate (S)-Et 2-ethoxy-3-(4-hydroxyphenyl)propionate (Ph3P, DEAD, THF) in 19% yield (2 steps). The intermediate ester was sapond. to give II. II had EC50 = 3.1 .mu.M for PPAR.alpha. and EC50 = 0.72 .mu.M for PPAR.gamma.. In vitro activation for PPAR.alpha./PPAR.gamma. was also detd. Claimed is a method for the treatment of obesity and diabetes.

IT **352286-24-9P 352286-26-1P**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); RCT (Reactant); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); RACT (Reactant or reagent); USES (Uses)
(synthesis of aryl-alkenyl-oxy-arylpropionic acid derivs. and their use in treatment of PPAR mediated disorders including **diabetes** and obesity)

IT **352286-25-0P 352286-27-2P**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(synthesis of aryl-alkenyl-oxy-arylpropionic acid derivs. and their use in treatment of PPAR mediated disorders including **diabetes** and obesity)

IT **5031-78-7 54916-28-8**
RL: RCT (Reactant); RACT (Reactant or reagent)
(synthesis of aryl-alkenyl-oxy-arylpropionic acid derivs. and their use in treatment of PPAR mediated disorders including **diabetes** and obesity)

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 5 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:359750 HCAPLUS
DOCUMENT NUMBER: 134:348284
TITLE: Phenyl compounds to treat diabetes and associated conditions
INVENTOR(S): Neogi, Partha; Nag, Bishwajit; Lakner, Frederick J.; Dey, Debendranath; Medicherla, Satyanarayana
PATENT ASSIGNEE(S): Calyx Therapeutics, Inc., USA
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2001034094 | A2 | 20010517 | WO 2000-US30927 | 20001108 |

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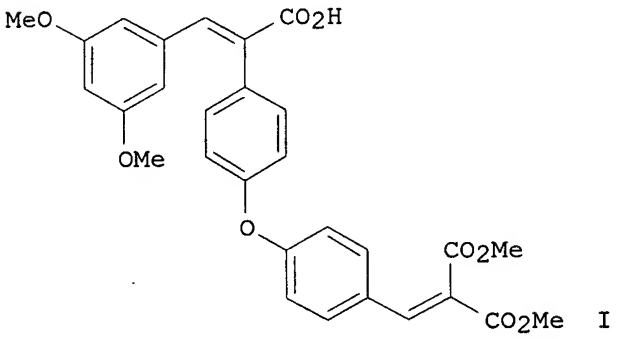
YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

AU 2001017607 A5 20010606 AU 2001-17607 20001108
PRIORITY APPLN. INFO.: US 1999-436047 A 19991108
WO 2000-US30927 W 20001108

OTHER SOURCE(S): MARPAT 134:348284
GI




I

AB Ph compds. (Markush included) are provided that lower blood glucose concns., lower serum triglyceride concns., lower systolic blood pressure, and increase glucose uptake by adipose tissue, but do not affect the expression of PPAR-.gamma. by adipose tissue. Compds. of the invention include e.g. I.

IT **339332-56-8 339332-57-9**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(Ph compds. to treat **diabetes** and assocd. conditions)

L12 ANSWER 6 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:22778 HCAPLUS
DOCUMENT NUMBER: 132:288524
TITLE: Effect of antihypertensive treatment on some hematological and biochemical parameters in japanese quails I-serum parameters
AUTHOR(S): Helal, Eman G. E.; Zaakhouk, Samir A. M.; El-Hakim, N. F. Abd
CORPORATE SOURCE: Department of Zoology, Faculty of Sciences, Al-Azhar University for Girls, Cairo, Egypt
SOURCE: Al-Azhar Bulletin of Science (1998), 9(1), 237-248
CODEN: ABSCE7; ISSN: 1110-2535
PUBLISHER: Al-Azhar University, Faculty of Science
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The present study aimed to investigate the mode of action of the antihypertensive agent captopril, which acts as angiotensin converting enzyme (ACE) inhibitor in animal bodies. These results showed the effect of the ACE inhibitor, captopril, on rectal temp., respiration rate, red blood cells count (R.B.Cs), Hb concn. (Hb%), and hematocrite (Hct) value. Also studies were conducted to examine serum glucose, total protein, albumin, total lipids, cholesterol level, and AST, ALT, LDH and T3 activities. Male and female (8 wk old) Japanese quail were daily treated with captopril in two doses (0.7 and 1.4 mg/kg body wt.), for 10 consecutive days and half of them were left for another 10 days for recovery. The results revealed a significant decrease in respiration rate, serum glucose, AST, ALT, total lipids and cholesterol of male and female Japanese quails. Captopril induced a significant decrease in albumin of male Japanese quail. Also significant changes were obsd. in Hct, serum globulin, and LDH. On the other hand, non-significant change was found in rectal temp., total protein and LDH in male only. Results also exhibited that there were different responses to captopril among male and female Japanese quails.

IT **6893-02-3**, t3
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**antihypertensive** treatment effect on some hematol. and biochem. parameters)

REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 7 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:795634 HCAPLUS
DOCUMENT NUMBER: 132:30840
TITLE: KB 285 in treatment of diabetes
INVENTOR(S): Apelqvist, Theresa; Efendic, Suad
PATENT ASSIGNEE(S): Karo Bio AB, Swed.
SOURCE: PCT Int. Appl., 20 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 9963976 | A2 | 19991216 | WO 1999-IB1175 | 19990607 |
| WO 9963976 | A3 | 20011220 | | |
| W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | | |
| RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG | | | | |
| AU 9941606 | A1 | 19991230 | AU 1999-41606 | 19990607 |
| EP 1143948 | A2 | 20011017 | EP 1999-925232 | 19990607 |

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI

PRIORITY APPLN. INFO.:

| | | |
|---|---|---|
| GB 1998-12314 | A | 19980608 |
| GB 1998-15149 | A | 19980713 |
| WO 1999-IB1175 | W | 19990607 |

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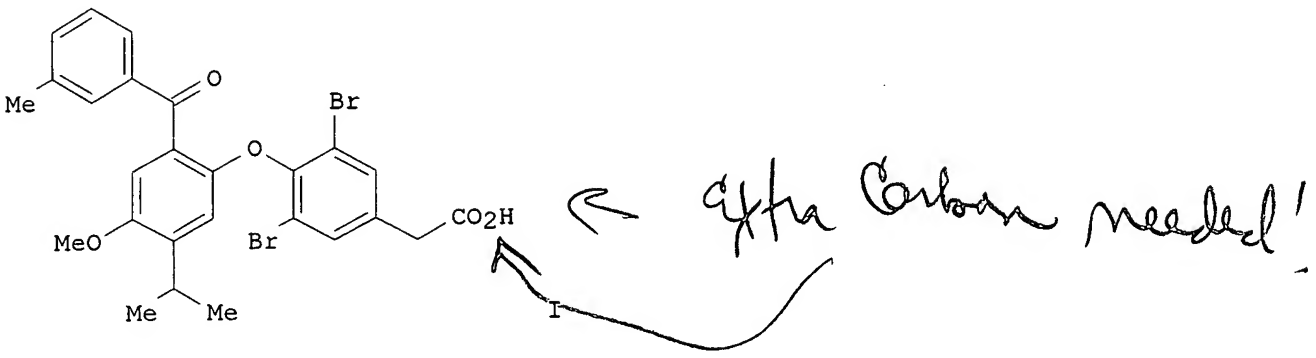



AB A liver-selective glucocorticoid antagonist, preferably KB285 (I) is prepd. and used in the prepn. of a pharmaceutical compns. for the treatment of diabetes. In addn. to synthetic examples, receptor binding and cell based assays are given.

IT **252201-98-2P**, KB 285
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(KB 285 in treatment of **diabetes**)

IT **252043-61-1P 252043-62-2P**
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(KB 285 in treatment of **diabetes**)

L12 ANSWER 8 OF 69 HCAPLUS COPYRIGHT 2002 ACS

| | |
|---|---|
| ACCESSION NUMBER: | 1999:736671 HCAPLUS |
| DOCUMENT NUMBER: | 131:351319 |
| TITLE: | Oxazolylmethoxybenzyl oxyiminoalkanoic acid derivatives with hypoglycemic and hypolipidemic activity |
| INVENTOR(S): | Momose, Yu; Odaka, Hiroyuki; Imoto, Hiroshi; Kimura, Hiroyuki; Sakamoto, Junichi |
| PATENT ASSIGNEE(S): | Takeda Chemical Industries, Ltd., Japan |
| SOURCE: | PCT Int. Appl., 148 pp.<br>CODEN: PIXXD2 |
| DOCUMENT TYPE: | Patent |
| LANGUAGE: | English |
| FAMILY ACC. NUM. COUNT: | 1 |

PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|

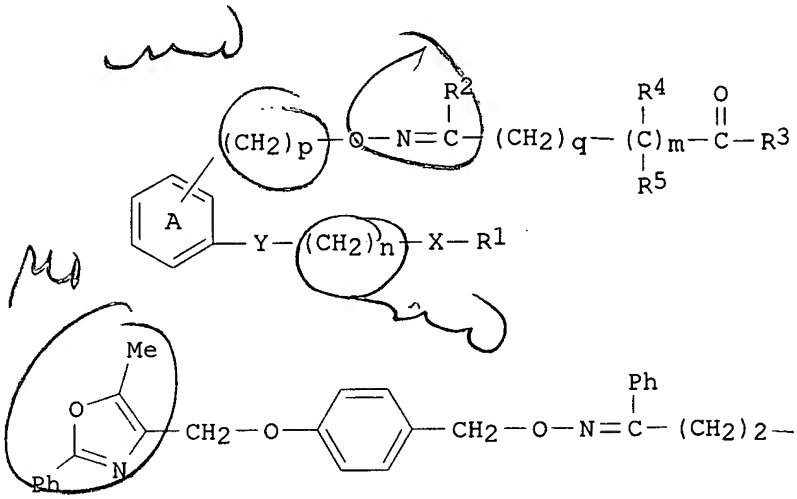
| | | | | | |
|---|---|---|---|---|---|
| WO 9958510 | A1 | 19991118 | WO 1999-JP2407 | 19990510 | |
| W: AE, AL, AM, AU, AZ, BA, BB, BG, BR, BY, CA, CN, CU, CZ, EE, GD, GE, HR, HU, ID, IL, IN, IS, JP, KG, KR, KZ, LC, LK, LR, LT, LV, MD, MG, MK, MN, MX, NO, NZ, PL, RO, RU, SG, SI, SK, SL, TJ, TM, TR, TT, UA, US, UZ, VN, YU, ZA, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | | | |
| RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG | | | | | |
| AU 9936297 | A1 | 19991129 | AU 1999-36297 | 19990510 | |
| BR 9910371 | A | 20010109 | BR 1999-10371 | 19990510 | |
| EP 1077957 | A1 | 20010228 | EP 1999-918355 | 19990510 | |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI | | | | | |
| JP 2000034266 | A2 | 20000202 | JP 1999-130543 | 19990511 | |
| JP 3074532 | B2 | 20000807 | | | |
| JP 2000198772 | A2 | 20000718 | JP 1999-373202 | 19990511 | |
| US 6251926 | B1 | 20010626 | US 1999-423854 | 19991115 | |
| LV 12606 | B | 20010520 | LV 2000-148 | 20001101 | |
| NO 2000005531 | A | 20010105 | NO 2000-5531 | 20001102 | |

PRIORITY APPLN. INFO.:

| | | |
|---|---|---|
| JP 1998-127921 | A | 19980511 |
| JP 1998-127922 | A | 19980511 |
| WO 1999-JP2407 | W | 19990510 |
| JP 1999-130543 | A3 | 19990511 |

OTHER SOURCE(S): MARPAT 131:351319


GI



I



II


AB Title compds. (I) [where R1 = (un)substituted hydrocarbon or heterocyclic group; X = bond, CO, CH(OH), or (alkyl)amino; n = 1-3; Y = O, S, SO, SO2, or (alkyl)amino; ring A = optionally substituted with 1-3 substituents; p = 1-8; R2 = H or (un)substituted hydrocarbon or heterocyclic group; q = 0-6; m = 0 or 1; R3 = OH, alkoxy, or (un)substituted NH2; R4 and R5 = independently H, hydrocarbon, or may form a ring with R2] were prepd. for the prevention or treatment of diabetes mellitus, hyperlipemia, insulin

insensitivity, insulin resistance, and impaired glucose tolerance. Thus, reaction of Me (E)-4-hydroxyimino-4-phenylbutyrate (prepn. given) with 4-(4-chloromethylphenoxymethyl)-5-methyl-2-phenyloxazole (prepn. given) in DMF followed by deesterification yielded (E)-II (60%). Representative compds. including II were mixed with a powdery diet and fed freely to KKAy mice for 4 days. Anal. of blood samples revealed 36% to 54% hypoglycemic action and 35% to 82% hypotriglyceridemic action of the treatment group compared to control animals. Compds. of the invention also exhibited excellent PPAR.gamma.-RXR.alpha. heterodimer ligand activity with EC50 values ranging from 0.024 .mu.M to 0.79 .mu.M.

IT **62936-33-8P 250602-60-9P 250602-61-0P 250602-79-0P**
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(intermediate; prepn. of oxazolylmethoxybenzyl oxyiminoalkanoic acid derivs. with hypoglycemic and hypolipidemic activity for treatment of **diabetes** mellitus and related conditions)

IT **250601-44-6P**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); RCT (Reactant); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); RACT (Reactant or reagent); USES (Uses)
(target compd.; prepn. of oxazolylmethoxybenzyl oxyiminoalkanoic acid derivs. with hypoglycemic and hypolipidemic activity for treatment of **diabetes** mellitus and related conditions)

IT **250601-08-2P 250601-09-3P 250601-45-7P**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(target compd.; prepn. of oxazolylmethoxybenzyl oxyiminoalkanoic acid derivs. with hypoglycemic and hypolipidemic activity for treatment of **diabetes** mellitus and related conditions)

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 9 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:412491 HCAPLUS
DOCUMENT NUMBER: 131:165733
TITLE: Leptin restores euglycemia and normalizes glucose turnover in insulin-deficient diabetes in the rat
AUTHOR(S): Chinookoswong, Narumol; Wang, Jin-Lin; Shi, Zhi-Qing
CORPORATE SOURCE: Department of Pharmacology, Amgen Center, Thousand Oaks, CA, USA
SOURCE: Diabetes (1999), 48(7), 1487-1492
CODEN: DIAEAZ; ISSN: 0012-1797
PUBLISHER: American Diabetes Association
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Leptin has been shown to improve insulin sensitivity and glucose metab. in normoinsulinemic healthy or obese rodents. It has not been detd. whether leptin may act independently of insulin in regulating energy metab. in vivo. The present study was designed to examine the effects of leptin treatment alone on glucose metab. in insulin-deficient streptozotocin (STZ)-induced diabetic rats. Four groups of STZ-induced diabetic rats

were studied: (1) rats treated with recombinant methionine murine leptin s.c. infusion with osmotic pumps for 12-14 days (LEP; 4 mg/kg/day); (2) control rats infused with vehicle (phosphate-buffered saline) for 12-14 days (VEH); (3) pair-fed control rats given a daily food ration matching that of LEP rats for 12-14 days (PF); and (4) rats treated with s.c. phloridzin for 4 days (PLZ; 0.4 g/kg twice daily). Phloridzin treatment normalizes blood glucose without insulin and was used as a control for the effect of leptin in correcting hyperglycemia. All animals were then studied with a hyperinsulinemic-euglycemic clamp (6 mU/kg/min). The authors' study demonstrates that leptin treatment in the insulin-deficient diabetic rats restored euglycemia, minimized body wt. loss due to food restriction, substantially improved glucose metabolic rates during the postabsorptive state, and restored insulin sensitivities at the levels of the liver and the peripheral tissues during the glucose clamp. The effects on glucose turnover are largely independent of food restriction and changes in blood glucose concn., as evidenced by the minimal improvement of insulin action and glucose turnover parameters in the PF and PLZ groups. The authors' results suggest that the antidiabetic effects of leptin are achieved through both an insulin-independent and an insulin-sensitizing mechanism.

IT **51-48-9**, T4, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(leptin **antidiabetic** effect mediation by insulin-independent and insulin-sensitizing mechanisms)

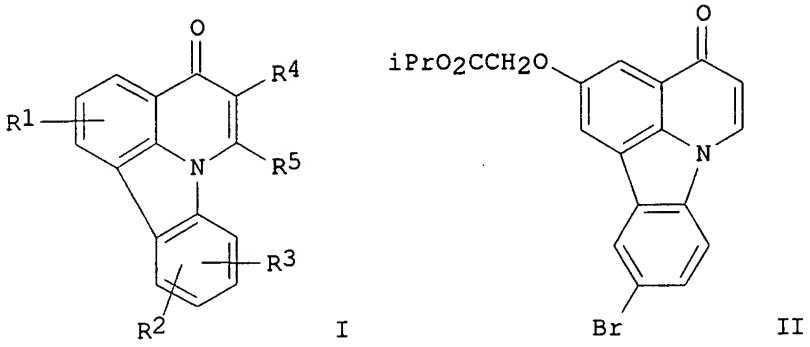
REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 10 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:355774 HCAPLUS
DOCUMENT NUMBER: 131:19021
TITLE: Preparation of pyridocarbazole derivatives with cyclic guanosine 3',5'-monophosphate-phosphodiesterase (cGMP-PDE) inhibitory activity
INVENTOR(S): Ohashi, Masayuki; Shudo, Toshiyuki; Nishijima, Kazumi; Notsu, Tatsuto; Kikuchi, Akira; Yanagibashi, Kazutoshi; Nishida, Hidemitsu
PATENT ASSIGNEE(S): Mochida Pharmaceutical Co., Ltd., Japan
SOURCE: PCT Int. Appl., 287 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 9926946 | A1 | 19990603 | WO 1997-JP4307 | 19971126 |

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR,

GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG
AU 9850670 A1 19990615 AU 1998-50670 19971126
PRIORITY APPLN. INFO.: WO 1997-JP4307 19971126
OTHER SOURCE(S): MARPAT 131:19021
GI




AB Novel pyridocarbazole derivs. [I; R1 = H, halo, cyano, (un)protected CO2H or carboxymethyl, C1-4 alkoxycarbonyl, CONH2, acetylamino, 3-carboxy-1-propenyl, 2-hydroxypentyloxy, 2,2-diethoxyethoxy, (un)protected OH or SH, linear or branched C1-4 alkanoyloxy, phenylcarbonyloxy, pyridylcarbonyloxy, optionally HO-substituted linear or branched alkyl C1-4 alkyl, optionally one or two alkyl-substituted NH2, (un)substituted C1-3 alkylthio, etc.; R2 = H, halo, (un)protected OH, SH, or NH2, cyano, NO2, CF3, CF3O, (un)protected CO2H, 4-morpholinylacetyl, linear or branched C1-4 alkanoyloxy, alkanoyl, or alkyl, (un)substituted C1-3 alkylthio, optionally C1-4 alkoxycarbonyl-substituted linear or branched C1-4 alkoxy; R3 = H, halo, (un)protected OH, linear or branched C1-4 alkoxy; R4 = H, halo, (un)protected CO2H, PhO, anilino, N-methylanilino, 4-morpholinylcarbonyl, optionally C3-6 cycloalkyl-substituted C1-2 alkyl, (un)substituted CH2Ph, optionally C1-4 alkyl-substituted pyridylmethyl, morpholinylmethyl, triazolylmethyl, furylmethyl, thienylmethyl, pyrimidinylmethyl, pyrazinylmethyl, pyrrolylmethyl, imidazolylmethyl, etc.; R5 = H, Me; provided that when R1 = R2 = R3 = R5 = H, R4 .noteq. H, CH2Ph, 4-diethylaminobenzyl, or furylmethyl] are prepd. These compds. have a highly selective inhibitory action on type-V and/or type-III cyclic GMP-phosphodiesterase. Also claimed are preventives and/or therapeutic agents for pulmonary hypertension, ischemic heart diseases, and diseases for which cGMP-PDE inhibition is effective, characterized by comprising at least one of the derivs. I as the active ingredient. Thus, 10-bromo-2-hydroxy-4H-pyrido[3,2,1-jk]carbazol-4-one was suspended in DMSO, stirred with K2CO3 at room temp. for 30 min and then with iso-Pr bromoacetate in the presence of KI at room temp. for 12 h to give the title compd. (II). II in vitro showed IC50 of 0.0008, >30, and >30 .mu.M against type V, III, and I PDE, resp. Pharmaceutical formulations contg. I were described.

IT **23689-01-2P**, 4-Acetyl-4'-methoxydiphenylamine
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT

(Reactant or reagent)
(prepn. of pyridocarbazole derivs. as cyclic guanosine monophosphate-phosphodiesterase (cGMP-PDE) inhibitors for treatment and prevention of pulmonary **hypertension** and ischemic heart disease)

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 11 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:270816 HCAPLUS
DOCUMENT NUMBER: 131:53579
TITLE: Inhibitory effects of tetrandrine and related synthetic compounds on angiogenesis in streptozotocin-diabetic rodents
AUTHOR(S): Kobayashi, Shinjiro; Kimura, Ikuko; Fukuta, Mizuki; Kontani, Hitoshi; Inaba, Kazuhiko; Niwa, Masashi; Mita, Shiro; Kimura, Masayasu
CORPORATE SOURCE: Department of Pharmacology, Faculty of Pharmaceutical Sciences, Hokuriku University, Kanazawa, 920-1181, Japan
SOURCE: Biological & Pharmaceutical Bulletin (1999), 22(4), 360-365
CODEN: BPBLEO; ISSN: 0918-6158
PUBLISHER: Pharmaceutical Society of Japan
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Structure-activity relationships of tetrandrine, isolated from a Kampo medicine, Stephania tetrandrae S. Moore (root), and related synthetic compds., were investigated in in vitro fetal bovine serum (FBS)-stimulated angiogenesis of cultured choroids in streptozotocin-diabetic Wistar rats, and air-pouch granuloma angiogenesis in vivo in diabetic mice. Tetrandrine, KS-1-1 (6,7-dimethoxy-1-[[4-[5-(6,7-dimethoxy-2-methyl-1,2,3,4-tetrahydroisoquino linyl)methyl-2-methoxy]phenoxy]benzyl]-2-methyl-1,2,3,4-tetrahydroisoquino line), and KS-1-4 (6,7-dimethoxy-1-[[4-[4-(6,7-dimethoxy-2-methyl-1,2,3,4-tetrahydroisoquino linyl)methyl]phenoxy]benzyl]-2-methyl-1,2,3,4-tetrahydroisoquinoline), potently inhibited choroidal angiogenesis and air-pouch granuloma angiogenesis in the diabetic state. Their inhibitory effects on diabetic choroids were greater than those on normal choroids. Among these compds., KS-1-4 inhibited only diabetic angiogenesis. These compds. significantly inhibited FBS-stimulated tube formation in vascular endothelial cells from normal rats. Tetradrine and KS-1-4, but not KS-1-1, inhibited vascular endothelial growth factor- and platelet-derived growth factor-BB-stimulated angiogenesis in normal choroids. The bis[tetrahydroisoquinoline] moiety, connected by oxy-bis[phenylenemethylene] and 2,2'-dimethyl groups in tetrandrine, contributes to the inhibition of diabetic choroidal angiogenesis. KS-1-4 may be a candidate for anti-choroidopathy and retinopathy drugs in the diabetic state.

IT **52533-03-6 228271-21-4 228271-23-6 228271-24-7 228271-26-9**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(inhibitory effects of tetrandrine and related synthetic compds. on angiogenesis in streptozotocin-**diabetic** rodents)

REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 12 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:208426 HCAPLUS
DOCUMENT NUMBER: 131:39512
TITLE: Alterations of heart function and Na+-K+-ATPase activity by etomoxir in diabetic rats
AUTHOR(S): Kato, Kiminori; Chapman, Donald C.; Rupp, Heinz; Lukas, Anton; Dhalla, Naranjan S.
CORPORATE SOURCE: Institute of Cardiovascular Sciences, St. Boniface General Hospital Research Centre, and Department of Physiology, Faculty of Medicine, University of Manitoba, Winnipeg, MB, R2H 2A6, Can.
SOURCE: Journal of Applied Physiology (1999), 86(3), 812-818
CODEN: JAPHEV; ISSN: 8750-7587
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB To examine the role of changes in myocardial metab. in cardiac dysfunction in diabetes mellitus, rats were injected with streptozotocin (65 mg/kg body wt) to induce diabetes and were treated 2 wk later with the carnitine palmitoyltransferase inhibitor (carnitine palmitoyltransferase I) etomoxir (8 mg/kg body wt) for 4 wk. Untreated diabetic rats exhibited a redn. in heart rate, left ventricular systolic pressure, and pos. and neg. rate of pressure development and an increase in end-diastolic pressure. The sarcolemmal Na+-K+-ATPase activity was depressed and was assocd. with a decrease in maximal d. of binding sites (Bmax) value for high-affinity sites for [3H]ouabain, whereas Bmax for low-affinity sites was unaffected. Treatment of diabetic animals with etomoxir partially reversed the depressed cardiac function with the exception of heart rate. The high serum triglyceride and free fatty acid levels were reduced, whereas the levels of glucose, insulin, and 3,3',-5-triiodo-L-thyronine were not affected by etomoxir in diabetic animals. The activity of Na+-K+-ATPase expressed per g heart wt., but not per mg sarcolemmal protein, was increased by etomoxir in diabetic animals. Furthermore, Bmax (per g heart wt) for both low-affinity and high-affinity binding sites in control and diabetic animals was increased by etomoxir treatment. Etomoxir treatment also increased the depressed left ventricular wt. of diabetic rats and appeared to increase the d. of the sarcolemma and transverse tubular system to normalize Na+-K+-ATPase activity. Therefore, a shift in myocardial substrate utilization may represent an important signal for improving the depressed cardiac function and Na+-K+-ATPase activity in diabetic rat hearts with impaired glucose utilization.
IT **6893-02-3**, 3,3',-5-Triiodo-L-thyronine
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(etomoxir effect on **diabetes**-induced alterations in sarcolemmal Na+-K+-ATPase, plasma lipids, and heart function)
REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 13 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:509103 HCAPLUS

DOCUMENT NUMBER: 129:156944
TITLE: Method for treating acid lipase deficiency diseases with a microsomal triglyceride transfer protein (MTP) inhibitor and cholesterol lowering drug
INVENTOR(S): Gregg, Richard E.; Wetterau, John R., II
PATENT ASSIGNEE(S): Bristol-Myers Squibb Co., USA
SOURCE: PCT Int. Appl., 61 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 9831367 | A1 | 19980723 | WO 1998-US619 | 19980113 |
| W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | | |
| RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG | | | | |
| US 6066653 | A | 20000523 | US 1998-5437 | 19980110 |
| AU 9861315 | A1 | 19980807 | AU 1998-61315 | 19980113 |

PRIORITY APPLN. INFO.: US 1997-36183P P 19970117
WO 1998-US619 W 19980113
OTHER SOURCE(S): MARPAT 129:156944
AB A method is provided for inhibiting or treating diseases assocd. with acid lipase deficiency by administering to a patient an MTP inhibitor, alone or optionally, in combination with another cholesterol lowering drug, e.g. pravastatin.
IT **51-49-0**, Dextrothyroxine **137-53-1**, Sodium dextrothyroxine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(acid lipase deficiency disease treatment with microsomal **triglyceride** transfer protein inhibitor and cholesterol lowering **drug**)

L12 ANSWER 14 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:509064 HCAPLUS
DOCUMENT NUMBER: 129:144862
TITLE: Method for treating or inhibiting phytosterolemia with a microsomal triglyceride transfer protein (MTP) inhibitor and cholesterol lowering drug
INVENTOR(S): Gregg, Richard E.
PATENT ASSIGNEE(S): Bristol-Myers Squibb Co., USA
SOURCE: PCT Int. Appl., 62 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 9831225 | A1 | 19980723 | WO 1998-US618 | 19980113 |
| W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | | |
| RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG | | | | |
| US 6057339 | A | 20000502 | US 1998-5430 | 19980110 |
| AU 9860232 | A1 | 19980807 | AU 1998-60232 | 19980113 |
| PRIORITY APPLN. INFO.: | | | US 1997-35591P P | 19970117 |
| | | | WO 1998-US618 W | 19980113 |

OTHER SOURCE(S): MARPAT 129:144862

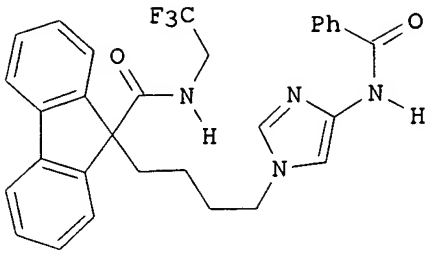
AB A method is provided for inhibiting onset or treating phytosterolemia by administering to a patient an MTP inhibitor, alone or, optionally, in combination with another cholesterol lowering drug, e.g. pravastatin.

IT **51-49-0**, Dextrothyroxine **137-53-1**, Sodium dextrothyroxine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(phytosterolemia treatment with microsomal **triglyceride** transfer protein inhibitor and cholesterol lowering **drug**)

L12 ANSWER 15 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:372652 HCAPLUS
DOCUMENT NUMBER: 129:54368
TITLE: Preparation of 9-heterocyclylalkyl-9-fluorenecarboxamides and analogs as microsomal triglyceride transfer protein inhibitors
INVENTOR(S): Biller, Scott A.; Dickson, John K.; Lawrence, R. Michael; Magnin, David R.; Poss, Michael A.; Robl, Jeffrey A.; Slusarchyk, William A.; Sulsky, Richard B.; Tino, Joseph A.
PATENT ASSIGNEE(S): USA
SOURCE: U.S., 240 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| US 5760246 | A | 19980602 | US 1996-767923 | 19961217 |

OTHER SOURCE(S): MARPAT 129:54368
GI



I

AB Title compds., e.g., R1Z1BCOAZ2R2 [A = bond, O, (alkyl)imino; B = e.g., C(ZR)2 in which RR = bond, O, NH, alk(en)ylene, etc., and Z = (un)substituted 1,2-phenylene; R1 = H, alk(en)yl, (hetero)aryl, etc.; R1 = groups cited for R1, haloalkyl, etc.; Z1 = (oxo- or aza)(oxo)alk(en)ylene, etc.; Z2 = bond, groups cited forZ1, etc.] were prepd. as microsomal triglyceride transfer protein inhibitors (no data). Thus, 9-fluorenecarboxylic acid was alkylated by Br(CH2)4Br and the CF3CH2NH2-amidated product arylated by 4-nitroimidazole to give, after redn. and N-acylation, title compd I.

IT **194209-66-0P 194210-59-8P 194211-14-8P**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. of 9-heterocyclylalkyl-9-fluorenecarboxamides and analogs as microsomal **triglyceride** transfer protein inhibitors)

L12 ANSWER 16 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:87580 HCAPLUS
DOCUMENT NUMBER: 128:162883
TITLE: Method for lowering serum lipid levels employing a microsomal triglyceride-transfer protein (MTP) inhibitor in combination with another cholesterol-lowering drug
INVENTOR(S): Gregg, Richard E.; Pouleur, Hubert G.; Wetterau, John R., II
PATENT ASSIGNEE(S): Bristol-Myers Squibb Co., USA
SOURCE: PCT Int. Appl., 60 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 9803069 | A1 | 19980129 | WO 1997-US12229 | 19970714 |

W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

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|---|---|---|---|---|
| ZA 9705950 | A | 19990104 | ZA 1997-5950 | 19970703 |
| AU 9736624 | A1 | 19980210 | AU 1997-36624 | 19970714 |
| AU 716145 | B2 | 20000217 | | |
| EP 1014791 | A1 | 20000705 | EP 1997-933435 | 19970714 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI | | | | |
| JP 2000515526 | T2 | 20001121 | JP 1998-507023 | 19970714 |

PRIORITY APPLN. INFO.: US 1996-22866P P 19960724
WO 1997-US12229 W 19970714

OTHER SOURCE(S): MARPAT 128:162883

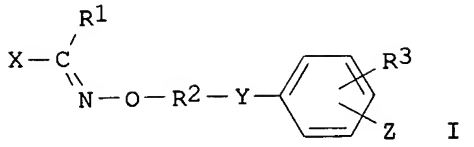
AB A method is provided for lowering serum lipids, cholesterol, and/or triglycerides and thereby inhibiting atherosclerosis by administering to a patient an MTP inhibitor in combination with a cholesterol lowering drug, e.g. pravastatin.

IT **51-49-0**, Dextrothyroxine **137-53-1**, Sodium Dextrothyroxine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(microsomal **triglyceride**-transfer protein (MTP) inhibitor combination with cholesterol-lowering **drug** for lowering serum lipid level)

L12 ANSWER 17 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:809855 HCAPLUS
DOCUMENT NUMBER: 128:123813
TITLE: Pharmaceuticals containing O-substituted oximes as blood sugar lowering agents.
INVENTOR(S): Yagisawa, Hiroaki; Fujita, Takeshi; Fujimoto, Koichi; Yoshioka, Takao; Wada, Kunio; Oguchi, Minoru; Fujiwara, Toshihiko; Horikoshi, Hiroyoshi
PATENT ASSIGNEE(S): Sankyo Co., Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 134 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| JP 09323929 | A2 | 19971216 | JP 1997-79691 | 19970331 |

PRIORITY APPLN. INFO.: JP 1996-79839 19960402
OTHER SOURCE(S): MARPAT 128:123813
GI



I

AB Title compds. I [R1 = H, C1-6 alkyl; R2 = C2-6 alkylene; R3 = H, C1-6 alkyl, etc.; X = (.alpha.-substituted) aryl; Y = O, S, NR4; R4 = H, C1-6 alkyl, C1-8 aryl; Z = dioxothiazolidinylidenemethyl, dioxothiazolidinylmethyl, dioxooxathiazolidinylmethyl, etc.], having blood sugar lowering activity among other biol. activities, are prepd. Thus, 2-[(benzylideneamino)oxy]ethanol (prepn. given) was reacted with 5-(p-hydroxybenzyl)-3-tritylthiazolidine-2,4-dione in THF contg. Ph3P and di-Et azodicarboxylate to give 5-[p-[2-(benzylideneaminooxy)ethoxy]benzyl]-3-tritylthiazolidine-2,4-dione, which was heated at 80.degree. in aq. dioxane contg. HOAc to give the title compd. I [R1 = R3 = H, R2 = (CH2)2, X = Ph, Y = O, Z = p-(2,4-dioxothiazolidin-5-ylmethyl)]. I [R1 = R3 = H, R2 = (CH2)2, X = 1-naphthyl, Y = O, Z = p-(2,4-dioxothiazolidin-5-ylmethyl)], also prepd., at 1 mg/Kg p.o. decreased the blood sugar level of hyperglycemic male mice by 22.1%. Pharmaceutical compns. contg. I are described.

IT **178054-52-9P**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); RCT (Reactant); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); RACT (Reactant or reagent); USES (Uses)
(**pharmaceuticals** contg. O-substituted oximes as **blood sugar** lowering agents)

IT **178054-53-0P**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(**pharmaceuticals** contg. O-substituted oximes as **blood sugar** lowering agents)

IT **10130-75-3**, 4'-(Phenylthio)acetophenone oxime **178056-18-3**, 4'-(Phenylsulfonyl)acetophenone oxime
RL: RCT (Reactant); RACT (Reactant or reagent)
(**pharmaceuticals** contg. O-substituted oximes as **blood sugar** lowering agents)

IT **178055-10-2P 178055-11-3P 178055-85-1P 178055-86-2P 178055-87-3P 178055-88-4P**
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(**pharmaceuticals** contg. O-substituted oximes as **blood sugar** lowering agents)

L12 ANSWER 18 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:737995 HCAPLUS
DOCUMENT NUMBER: 128:43714
TITLE: The modulatory effect of antidiabetic drugs on thyroid function in diabetic rats

AUTHOR(S): Abdel Ghany Nabila, Rasha H.; El-Maraghy, N.; Zakaria, Mohamed N. M.; Eid, Naglaa F.
CORPORATE SOURCE: Department of Pharmacology, Faculty of Pharmacy, Zagazig University, Egypt
SOURCE: Zagazig Journal of Pharmaceutical Sciences (1996), 5(2), 77-83
CODEN: ZJPSEV; ISSN: 1110-5089
PUBLISHER: University of Zagazig, Faculty of Pharmacy
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The effects of 30 days treatment with chlorpropamide (5 mg/kg/day), glipizide (2.5 mg/kg/day), and metformin (350 mg/kg/day), on blood glucose level, serum fructosamine, glycosylated Hb (HbA1), triiodothyronine (T3) and thyroxine (T4) in diabetic rats were investigated. Animals were randomly assigned into three equal groups, each group received a single daily dose of one of the previously mentioned antidiabetic drugs. The parameters of interest are demonstrated before, ten and thirty days after drug administration. In the present study, all the used drugs significantly reduced the blood glucose level after 10 and 30 days. Both fructosamine and HbA1 are significantly elevated after 30 days of chlorpropamide and glipizide administration, while the two parameters were non-significantly changed in the group received metformin. Triiodothyronin was significantly decreased in all groups after 30 days of treatment. It could be concluded that both chlorpropamide, glipizide, and metformin decreased the elevated blood glucose level in diabetic rats. The change in fructosamine and glycolated Hb was not indicative to the change in blood glucose level induced by glipizide, chlorpropamaide and metformin as the 30 days of study did not cover the required period needed for such correlation to occur. Triiodothyronine and thyroxin levels were decreased and increased resp. in diabetic rats treated by chlorpropamaide or glipizide indicating a modulatory action of such antidiabetic agents on the thyroid function. Metformin lowered T3 but showed a limited effect on T4 level.

IT **51-48-9**, Thyroxine, biological studies **6893-02-3**, Triiodothyronine
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**antidiabetic drugs** effect on thyroid function)

L12 ANSWER 19 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:684384 HCAPLUS
DOCUMENT NUMBER: 127:307307
TITLE: Preparation of phenylalkylcarboxylic acid derivatives lowering blood sugar level
INVENTOR(S): Yanagisawa, Hiroaki; Takamura, Makoto; Fujita, Takashi; Fujiwara, Toshihiko
PATENT ASSIGNEE(S): Sankyo Co., Ltd., Japan; Yanagisawa, Hiroaki; Takamura, Makoto; Fujita, Takashi; Fujiwara, Toshihiko
SOURCE: PCT Int. Appl., 339 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 9737970 | A1 | 19971016 | WO 1997-JP1122 | 19970401 |
| W: AU, CA, CN, CZ, HU, KR, MX, NO, NZ, RU, US | | | | |
| RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE | | | | |
| CA 2251468 | AA | 19971016 | CA 1997-2251468 | 19970401 |
| AU 9720446 | A1 | 19971029 | AU 1997-20446 | 19970401 |
| AU 708919 | B2 | 19990819 | | |
| EP 916651 | A1 | 19990519 | EP 1997-908566 | 19970401 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI | | | | |
| CN 1219927 | A | 19990616 | CN 1997-194978 | 19970401 |
| RU 2169141 | C2 | 20010620 | RU 1998-119843 | 19970401 |
| JP 09323967 | A2 | 19971216 | JP 1997-85076 | 19970403 |
| NO 9804633 | A | 19981203 | NO 1998-4633 | 19981002 |
| KR 2000005224 | A | 20000125 | KR 1998-7911 | 19981002 |
| US 6103907 | A | 20000815 | US 1998-168973 | 19981005 |

PRIORITY APPLN. INFO.: JP 1996-82803 A 19960404
WO 1997-JP1122 W 19970401

OTHER SOURCE(S): MARPAT 127:307307

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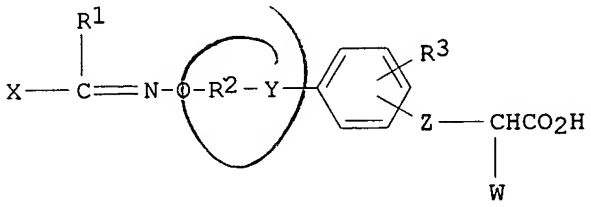

I

AB Phenylalkylcarboxylic acid derivs. represented by general formula [I; R1 = C1-6 linear or branched alkyl; R2 = C2-6 linear or branched alkylene; R3 = H, C1-6 linear or branched alkyl, C1-4 linear or branched alkoxy, C1-4 linear or branched alkylthio, halo, NO2, di(C1-4 linear or branched alkyl)amino, (un)substituted C6-10 aryl or C7-12 aralkyl; X = (un)substituted C6-10 aryl, 5- to 10-membered mono- or bicyclic arom. heterocyclyl contg. 1-4 heteroatoms selected from O, S, and N; Y = O, S, (un)substituted NH; Z = single bond, C1-6 linear or branched alkylene; W = C1-6 linear or branched alkyl, C1-4 linear or branched alkoxy, C1-4 linear or branched alkylthio, NH2, mono- or di(C1-4 linear or branched alkyl)amino, etc.] and pharmacol. acceptable salts and esters thereof, useful as a remedy or preventive for hyperglycemia and the like, are prepd. Thus, Et 2-ethoxy-3-(4-hydroxyphenyl)propionate was treated with NaH in DMF and PhMe under stirring at room temp. for 30 min and condensed with 4'-(2-pyridyl)acetophenone oxime O-2-(methanesulfonyloxyethyl) ether under stirring at 80.degree. for 3 h, followed by sapon. with 1N aq. NaOH and EtOH and acidification with 1N aq. HCl to give the title compd. (II; R = EtO). II (R = EtO) and II (R = EtNH) at 1 mg/kg p.o. lowered blood sugar by 21.9 and 26.9%, resp., in hyperglycemic mice. A capsule, a tablet, and a granule formulation contg. II (R = EtO) were prepd.

IT **197299-20-0P**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. of phenylalkylcarboxylic acid derivs. lowering **blood sugar** level)

L12 ANSWER 20 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:535299 HCAPLUS
DOCUMENT NUMBER: 127:233085
TITLE: Alterations of thyroid hormone in prognosis of diabetics
AUTHOR(S): Zhou, Yuan; Chen, Guangming
CORPORATE SOURCE: Department of Surgery, People's Hospital of Guannan County, Guannan, 223500, Peop. Rep. China
SOURCE: Jiangsu Yiyao (1997), 23(3), 171-176
CODEN: CIYADX; ISSN: 0253-3685
PUBLISHER: Jiangsu Yiyao Bianjibu
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB Serum thyroid hormones were obsd. in 78 patients with diabetics. Serum total and free T3 (triiodothyronine) of the patients were decreased, esp. those with uncontrolled blood glucose or with acute complication. Furthermore, IDDM had lower T3 than the NIDDM. The results suggest that decrease of serum T3 is helpful in prognosis evaluation of diabetics.
IT **6893-02-3**, Triiodothyronine
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); OCCU (Occurrence); USES (Uses)
(triiodothyronine level in blood serum in relation to human **diabetes** prognosis)

L12 ANSWER 21 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:499168 HCAPLUS
DOCUMENT NUMBER: 127:190649
TITLE: Preparation of 9-aralkyl-9-fluorenecarboxamides and analogs as microsomal triglyceride transfer protein inhibitors
INVENTOR(S): Biller, Scott A.; Dickson, John K.; Lawrence, R. Michael; Magnin, David R.; Poss, Michael A.; Robl, Jeffrey A.; Slusarchyk, William A.; Sulsky, Richard B.; Tino, Joseph A.
PATENT ASSIGNEE(S): Bristol-Myers Squibb Co., USA
SOURCE: PCT Int. Appl., 615 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 9726240 | A1 | 19970724 | WO 1997-US587 | 19970113 |

W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE,

ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

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|---|---|---|---|---|---|
| CA 2236684 | AA | 19970724 | CA 1997-2236684 | 19970113 | |
| AU 9718285 | A1 | 19970811 | AU 1997-18285 | 19970113 | |
| AU 716729 | B2 | 20000302 | | | |
| CN 1209803 | A | 19990303 | CN 1997-191713 | 19970113 | |
| EP 904262 | A1 | 19990331 | EP 1997-903805 | 19970113 | |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI | | | | | |
| BR 9707607 | A | 19990727 | BR 1997-7607 | 19970113 | |
| JP 2000502355 | T2 | 20000229 | JP 1997-526127 | 19970113 | |
| ZA 9700328 | A | 19970715 | ZA 1997-328 | 19970115 | |
| NO 9803268 | A | 19980715 | NO 1998-3268 | 19980715 | |

PRIORITY APPLN. INFO.:
US 1996-10346P P 19960116
US 1996-17224P P 19960509
US 1996-30370P P 19961105
WO 1997-US587 W 19970113

OTHER SOURCE(S): MARPAT 127:190649

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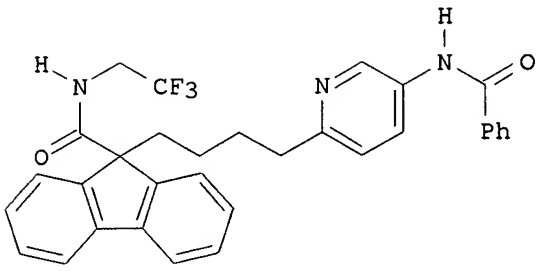

I

AB R2Z4Z3ZZ2Z1R1 [R1 = H, (cyclo)alk(en)yl, alkoxy, (hetero)aryl(oxy), etc.; R2 = groups cited for R1, haloalkyl, etc.; Z = CO, SO0-2, CR(OH); R = H, alkyl, aryl; Z1 = (O- or NH-interrupted)(oxo)alk(en)ylene, etc.; Z2 = (un)substituted 9H-fluoren-9-ylidene, 9H-xanthen-9-ylidene, etc.; Z3 = bon, O, NR5; R5 = H or alkyl; R2R5 = atoms to form a ring; Z4 = bond, groups cited for Z1] were prepd as microsomal triglyceride transfer protein inhibitors (no data). Thus, 9H-fluorene-9-carboxylic acid was alkylated by TsOCH2CH2C.tplbond.CH and the product amidated by H2NCH2CF3 9-(3-butynyl)-N-(2,2,2-trifluoroethyl)fluorene-9-carboxamide which was arylated by 2-bromo-5-nitropyridine to give, after redn. and BzCl amidation, title compd. I.

IT **194209-66-0P 194210-59-8P 194211-14-8P**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. of 9-aralkyl-9-fluorenecarboxamides and analogs as microsomal

**triglyceride** transfer protein inhibitors)

L12 ANSWER 22 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:128355 HCAPLUS
DOCUMENT NUMBER: 126:207671
TITLE: Maternal nonthyroidal illness and fetal thyroid hormone status, as studied in the streptozotocin-induced diabetes mellitus rat model
AUTHOR(S): Calvo, Rosa; Morreale de Escobar, Cabriella; Escobar del Ray, Francisco; Obregon, Maria-Jesus
CORPORATE SOURCE: Facultad de Medicina, University Autonoma de Madrid, Madrid, 28029, Spain
SOURCE: Endocrinology (1997), 138(3), 1159-1169
CODEN: ENDOAO; ISSN: 0013-7227
PUBLISHER: Endocrine Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We have used the streptozotocin-induced diabetes mellitus pregnant rat as a model of maternal nonthyroidal illness. We measured the effects of different degrees of diabetes mellitus on maternal body wt., the outcome of pregnancy, circulating glucose, insulin, T4, T3, rT3, and TSH in mother and fetus, T4 and T3 in maternal and fetal tissues, and iodothyronine deiodinases in liver, lung, and brain. All of the changes in thyroid hormone status typical of nonthyroidal illnesses were obsd. in the mothers and were related to the degree of the metabolic imbalances. Most were controlled with a daily insulin dose of 0.5 U/100 g BW. Normalization of maternal placental T4, however, required higher insulin doses than in other maternal tissues. The no. and body wt. of the fetuses, their pituitary GH contents, and their thyroid hormone status were severely affected. The total extrathyroidal T4 and T3 pools decreased to one third of normal fetal values. T4 and T3 concns. in the fetal brain were lower than normal, and the expected increase in type II 5'-deiodinase activity was not obsd. The low cerebral T3 only improved with adequate insulin treatment of the dams. It is concluded that maternal diabetes mellitus, and possibly other nonthyroid illnesses that impair the availability of intracellular energy stores, may affect fetal brain T3 when thyroid hormones are essential for normal development.
IT **51-48-9**, T4, biological studies **5817-39-0**, RT3 **6893-02-3**, T3
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(maternal nonthyroidal illness and impaired fetal thyroid hormone status, as studied in streptozotocin-induced **diabetes** mellitus rat model)

L12 ANSWER 23 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1996:686080 HCAPLUS
DOCUMENT NUMBER: 126:1499
TITLE: Effects of methimazole in the early and established phases of NG-nitro-L-arginine methyl ester hypertension
AUTHOR(S): Vargas, Felix; Fernandez-Rivas, Antonio; Osuna, Antonio
CORPORATE SOURCE: Fac. Med., Serv. Nefrologia, Granada, E-18012, Spain

SOURCE: European Journal of Endocrinology (1996), 135(4), 506-513
CODEN: EJOEEP; ISSN: 0804-4643
PUBLISHER: Scandinavian University Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB In the present study we evaluated the effects of methimazole, an antithyroid drug, on blood pressure and other variables in the early and established phases of hypertension induced by the inhibition of nitric oxide synthesis with the oral administration of NG-nitro-L-arginine Me ester (L-NAME), 75 mg/100 mL in the drinking water. Moreover, we also evaluated the acute pressor effect of L-NAME on systemic blood pressure in control and rats treated chronically with methimazole, administered via drinking water (30 mg/100 mL). Oral administration of methimazole maintained the blood pressure of L-NAME-treated rats at normal levels 25 days after induction of hypertension. However, after 25 days of methimazole treatment in rats made hypertensive with L-NAME (for 25 days), high blood pressure was similar in methimazole-treated and non-treated L-NAME rats, despite the fact that a hypothyroid state had been achieved in the methimazole-treated rats. Acute i.v. injection of L-NAME caused a similar increase in mean arterial pressure in control and methimazole-treated rats at the lowest dose; however, smaller pressor responses were obsd. with increasing doses in hypothyroid rats. These results clearly demonstrate that hypothyroidism induced by methimazole prevents, but does not reverse, L-NAME hypertension and reduces the acute pressor responsiveness to L-NAME administration.

IT **51-48-9**, T4 Hormone, biological studies **6893-02-3**, T3 Hormone
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(effects of methimazole in early and established phases of nitroarginine Me ester **hypertension**)

L12 ANSWER 24 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1996:379688 HCAPLUS
DOCUMENT NUMBER: 125:58495
TITLE: Oxime-containing thiazolidinedione derivatives and analogs, their preparation, and their therapeutic use against diabetes and related conditions.
INVENTOR(S): Yanagisawa, Hiroaki; Fujita, Takashi; Fujimoto, Koichi; Yoshioka, Takao; Wada, Kunio; Oguchi, Minoru; Fujiwara, Toshihiko; Horikoshi, Hiroyoshi
PATENT ASSIGNEE(S): Sankyo Co., Ltd., Japan
SOURCE: Eur. Pat. Appl., 252 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| EP 708098 | A1 | 19960424 | EP 1995-307131 | 19951009 |
| EP 708098 | B1 | 19990303 | | |

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE

| | | | | | | |
|---|---|---|---|---|---|---|
| CA | 2159938 | AA | 19960408 | CA | 1995-2159938 | 19951005 |
| JP | 09048779 | A2 | 19970218 | JP | 1995-258789 | 19951005 |
| JP | 2843281 | B2 | 19990106 | | | |
| US | 5703096 | A | 19971230 | US | 1995-539541 | 19951005 |
| FI | 9504763 | A | 19960408 | FI | 1995-4763 | 19951006 |
| NO | 9503990 | A | 19960409 | NO | 1995-3990 | 19951006 |
| ZA | 9508465 | A | 19960424 | ZA | 1995-8465 | 19951006 |
| AU | 9533076 | A1 | 19960502 | AU | 1995-33076 | 19951006 |
| AU | 688573 | B2 | 19980312 | | | |
| HU | 72642 | A2 | 19960528 | HU | 1995-2925 | 19951006 |
| CN | 1143639 | A | 19970226 | CN | 1995-119194 | 19951006 |
| CN | 1056840 | B | 20000927 | | | |
| RU | 2122998 | C1 | 19981210 | RU | 1995-117054 | 19951006 |
| IL | 115536 | A1 | 19990714 | IL | 1995-115536 | 19951006 |
| TW | 400329 | B | 20000801 | TW | 1995-84110541 | 19951006 |
| AT | 177088 | E | 19990315 | AT | 1995-307131 | 19951009 |
| ES | 2132536 | T3 | 19990816 | ES | 1995-307131 | 19951009 |
| US | 5780490 | A | 19980714 | US | 1997-878219 | 19970618 |
| US | 5972959 | A | 19991026 | US | 1998-63609 | 19980421 |

PRIORITY APPLN. INFO.:

| | | | |
|---|---|---|---|
| JP | 1994-243876 | A | 19941007 |
| JP | 1995-136788 | A | 19950602 |
| US | 1995-539541 | A3 | 19951005 |
| US | 1997-878219 | A3 | 19970618 |

OTHER SOURCE(S): MARPAT 125:58495

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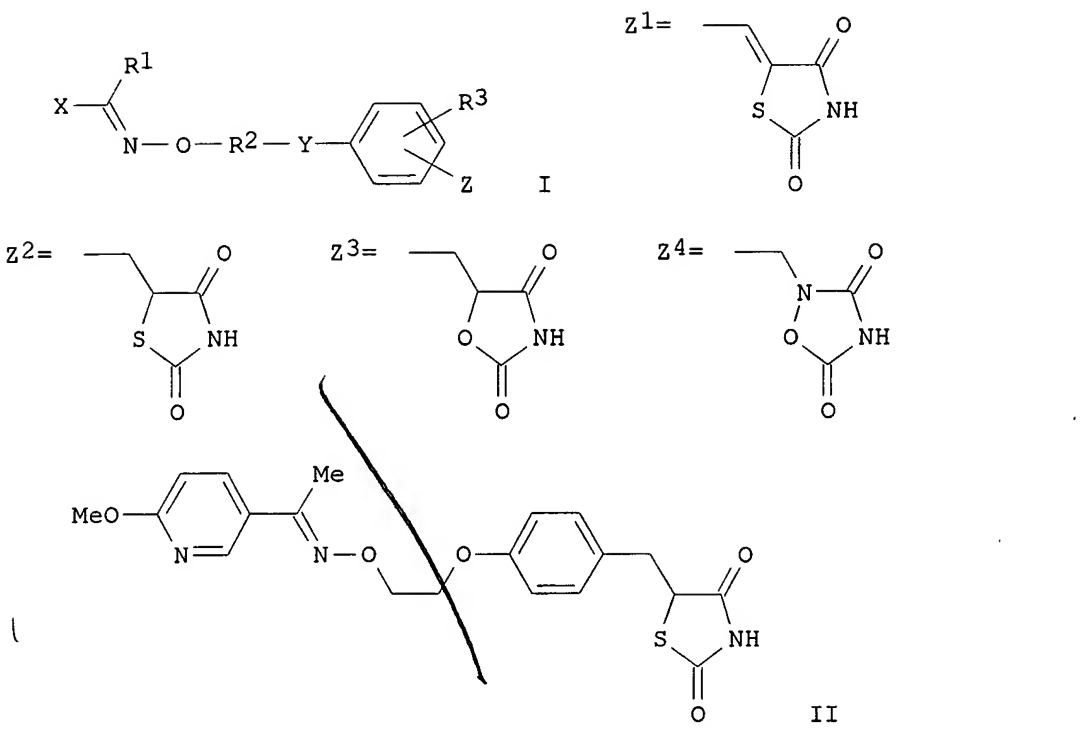


AB Title compds. I [R1 = H or alkyl; R2 = alkylene; R3 = H, alkyl, alkoxy, alkylthio, halo, NO2, (di)(alkyl)amino, aryl, or aralkyl; X = aryl or arom. heterocyclyl; Y = O, S, NR4; R4 = H, alkyl, acyl; Z = Z1-Z4] and salts were prepd. The compds. are useful for treating or preventing hyperlipidemia, hyperglycemia, obesity, impaired glucose tolerance (IGT), insulin resistant non-IGT (NGT), non-diagnostic GT, insulin resistance, diabetic complications, fatty liver, polycystic ovary syndrome (PCOS) and gestational diabetes mellitus (GDM); in addn., they have aldose reductase inhibitory activity (no data). For example, etherification of 2-[[[1-(2-methoxy-5-pyridyl)ethylidene]amino]oxy]ethanol with 5-(4-hydroxybenzyl)-3-tritylthiazolidine-2,4-dione by Mitsunobu reaction, and detritylation in aq. AcOH-dioxane at 80.degree., gave preferred title compd. II. At 1 mg/kg orally in hyperglycemic mice, II reduced blood glucose by 49.3% after 3 h.

IT **178055-10-2P 178055-11-3P 178055-85-1P 178055-86-2P 178055-87-3P 178055-88-4P**
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(intermediate; prepn. of oxime-contg. thiazolidinedione derivs. and analogs as **antidiabetics**)

IT **178054-52-9P 178054-53-0P**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. of oxime-contg. thiazolidinedione derivs. and analogs as

**antidiabetics**)
IT **10130-75-3 178056-18-3**
RL: RCT (Reactant); RACT (Reactant or reagent)
(starting material; prepn. of oxime-contg. thiazolidinedione derivs. and analogs as **antidiabetics**)

L12 ANSWER 25 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1996:326164 HCAPLUS
DOCUMENT NUMBER: 125:10826
TITLE: Preparation of p-[(phenoxy or benzyloxy)phenoxy]benzylazole derivatives for lowering blood sugar
INVENTOR(S): Niigata, Kunihiro; Takahashi, Takumi; Maruyama, Tatsuya; Suzuki, Takayuki; Onda, Kenichi; Konya, Tooru; Noshiro, Osamu
PATENT ASSIGNEE(S): Yamanouchi Pharma Co Ltd, Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 26 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| JP 08059638 | A2 | 19960305 | JP 1994-202503 | 19940826 |

OTHER SOURCE(S): MARPAT 125:10826
GI For diagram(s), see printed CA Issue.
AB The title compds. (I; ring A = imidazolyl, tetrazolyl, Q, Q1; wherein X = O, S, NH; Y = N, CH; R1 = H, halo, lower alkyl, lower hydroxyalkyl, lower alkoxy, CF3, NO2, CO2H, lower alkoxycarbonyl, CH2 NHCONHCO2R5, CH:NOH; wherein R5 = H, lower alkyl; R2, R3 = H, halo; R4 = H, HO; n = 0,1), which lower blood sugar based on the enhancement of insulin sensitivity, have low toxicity, and are useful as antidiabetics for treating or preventing noninsulin-dependent diabetes and various diabetes complications (no data), are prepd. Thus, 3-(4-trifluoromethylphenoxy)phenol 6, K2CO3 3.3, and 4-fluorobenzaldehyde 3.0 g were stirred in DMSO at 100.degree. for 10 h to give 6 g 4-[3-(4-trifluoromethylphenoxy)phenoxy]benzaldehyde (II; R = CHO), which (6 g) was condensed with 1.8 g hydroxylamine hydrochloride in the presence of 2.0 g NH4OAc in aq. MeOH at room temp. for 2 h and under reflux for 30 min to give the oxime II (R = CH:NOH) (4.0 g). The latter oxime (3.0 g) was dissolved in 30 mL EtOH and after adding 1.2 g pyridine-borane complex, treated dropwise with 12 mL 4 n aq. HCl, and left to stand at room temp. for 4 h to give 2.5 g II (R = CH2NHOH), which (1.5 g) was dissolved in THF, treated with 0.7 g ethoxycarbonyl isocyanate, left to stand for 30 min, made alk. with 1 N aq. NaOH, left to stand at room temp. for 2 h, and made acidic with 6 N aq. HCl to give 1.0 g the 1,2,4-oxadiazolidine-3,5-dione deriv. II (R = Q2).
IT **177031-98-0P**
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(prepn. of p-[(phenoxy or benzyloxy)phenoxy]benzylazole derivs. for lowering **blood sugar** as **antidiabetics**)

L12 ANSWER 26 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1995:882704 HCAPLUS
DOCUMENT NUMBER: 123:306032
TITLE: Effect of pazufloxacin on blood glucose levels in rats
AUTHOR(S): Kimura, Kazuyuki; Iwai, Masakazu; Taguchi, Masahiro; Hayashi, Hitofumi; Hanada, Shuichi; Koshiba, Hiroshi; Kawabata, Yoshiyasu; Hori, Seiji; Shimada, Jingoro
CORPORATE SOURCE: Inst. Med. Sci., St. Marianna Univ. Sch. Med., Kawasaki, 216, Japan
SOURCE: Nippon Kagaku Ryoho Gakkai Zasshi (1995), 43(Suppl. 2), 132-42
CODEN: NKRZE5
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB The effect of pazufloxacin (PZFX), a synthetic new quinolone antimicrobial agent, on blood glucose levels and several factors affected to them was evaluated in rats using single or 28-days repeated administration. In preliminary assessment of the evaluation model in rats, the assay system for blood glucose-regulating factors (insulin, etc.) was judged as reliable, since it produced results in an oral glucose loading test consistent with those reported elsewhere. It was found possible to narrow down diurnal variations by introducing fasting from 21:00 of the previous day. Human test kits used for the measurement of insulin and thyroid hormones were usable for the measurement of these parameters in the blood of rats. In the single administration test, PZFX reached satn. in the blood at 1200 mg/kg, which corresponded to 100 times the proposed clin. dose. However, no abnormal or specific changes were noted in any biochem. parameters (such as blood glucose, insulin, glucagon, and thyroid hormones etc.) or in histopathol. examn. of the pancreas (islets of Langerhans). In the repeated administration test, a dose of 600 mg/kg (.apprx.50 times the proposed clin. dose) repeated once daily for 28 days inhibited body wt. gain from the 2nd day until the end of administration. This dose was thus estd. to be high enough to induce toxicity. In the repeated administration test, insulin level tended to increase on the final day of administration. Blood glucose level underwent no change. This seemed to be a phenomenon not assocd. with the drug. No abnormal or specific changes were detected in other biochem. parameters or in histopathol. examn. of the pancreas (islets of Langerhans). The present test results combined with results of earlier toxicity tests suggest that it is unlikely that PZFX exerts the effect on blood glucose levels and blood glucose-regulating factors.
IT **51-48-9**, Thyroxine, biological studies **6893-02-3**, Triiodothyronine
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(no or little effect of pazufloxacin on **blood glucose** and **blood glucose**-regulating factors)

L12 ANSWER 27 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1995:864561 HCAPLUS
DOCUMENT NUMBER: 123:275649
TITLE: Effects of aspirin on blood glucose and hormonal levels in diabetic and DOCA-treated nephrectomized-hypertensive rats
AUTHOR(S): El-Fayoumi, Hassan M.; Zakaria, Mohamed N.M.; Gharieb,

Salah A.
CORPORATE SOURCE: Faculty of Pharmacy, Zagazig University, Egypt
SOURCE: Zagazig J. Pharm. Sci. (1995), Volume Date 1995, 4(1-B), 219-26
CODEN: ZJPSEV
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The effects of aspirin on blood glucose, lactate, cortisol and thyroid hormone levels in diabetic and DOCA-treated nephrectomized-hypertensive rats were investigated. The results showed that induction of diabetes in normotensive rats produced an elevation in blood glucose, cortisol and lactate levels by 160%, 26% and 82%, resp., while it reduced the thyroid hormonal (T3 & T4) levels to 86% and 73% of the normal values. In DOCA-treated nephrectomized-hypertensive rats the blood glucose and lactate levels were reduced to 80% and 56% compared with the universal control rats, while the blood levels of cortisol, T3 & T4 were non significantly affected. In alloxan-treated hypertensive rats there was no effect on the above mentioned parameters except T3 level which was decreased to 82% of the original value. Administration of aspirin significantly reduced the blood glucose and cortisol levels in diabetic (to 55% & 65%, resp.) hypertensive (to 93% & 73%, resp.) and alloxan-treated hypertensive rats (to 94% & 72%, resp.) compared with control group. The blood levels of lactate, T3 & T4 were not affected. Thus, the redn. produced by aspirin on both blood glucose and cortisol levels may be attributed in part to its ability for inhibition of prostaglandin synthesis which indirectly affect the insulin release from .beta.-cells.

IT **51-48-9**, Thyroxine, biological studies **6893-02-3**, Triiodothyronine
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(effects of aspirin on **blood glucose** and hormonal levels in **diabetic** and DOCA-treated nephrectomized-**hypertensive** rats)

L12 ANSWER 28 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1995:280330 HCAPLUS
DOCUMENT NUMBER: 122:46129
TITLE: Effect of chronic treatment with prazosin and L-arginine on the elevation of blood pressure during cold exposure
AUTHOR(S): Fregly, Melvin J.; Rossi, Fabian; Sun, Zhongjie; Tumer, Nihal; Cade, J. Robert; Hegland, Donald; Yurekli, Muhittin
CORPORATE SOURCE: Departments of Physiol., Pharmacol. Med., Univ. Florida, Gainesville, FL, USA
SOURCE: Pharmacology (1994), 49(6), 351-62
CODEN: PHMGBN; ISSN: 0031-7012
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Chronic exposure to cold (5.degree.C) is well known to increase both tyrosine hydroxylase (TH) activity in brown adipose tissue and systemic blood pressure. The effect of chronic dietary administration of the .alpha.-adrenergic antagonist, prazosin, and the amino acid, L-arginine, on both the elevation of blood pressure during exposure to cold and on TH

activity and expression of TH mRNA in the adrenal glands of rats was studied. As obsd. previously, chronic exposure to cold increased systolic blood pressure significantly and induced cardiac hypertrophy. Chronic dietary treatment with prazosin (8 mg/kg food) and arginine (20 g/kg food) returned blood pressure to control levels, did not affect body wt. significantly, but failed to prevent cardiac hypertrophy. Both prazosin and L-arginine reduced the drinking response to administration of angiotensin II. Treatment with arginine and prazosin was accompanied by a significant increase in the urinary outputs of dopamine and L-DOPA. The 3 cold-treated groups (control, L-arginine and prazosin) had increases in plasma T3 and decreases in plasma T4 and plasma renin activity. Plasma concns. of epinephrine and norepinephrine were increased significantly in the L-arginine-treated group. TH mRNA and TH activity in the adrenal glands were increased in the 3 cold-treated groups and these measures were correlated directly and significantly with plasma norepinephrine and epinephrine concns. Although both prazosin and arginine prevented the cold-induced elevation of blood pressure, they did not prevent the increase in TH mRNA, TH activity or epinephrine in plasma. The protective effect of arginine and prazosin in cold-induced hypertension may be related both to their redn. in plasma renin activity and to a reduced responsiveness to angiotensin II, as well as to their abilities to increase the secretion of dopamine.

IT **51-48-9**, Thyroxine, biological studies
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(prazosin and arginine prevention of chronic cold-induced **hypertension** in relation to effect on thyroxine)

IT **6893-02-3**, Triiodothyronine
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(prazosin and arginine prevention of chronic cold-induced **hypertension** in relation to effect on triiodothyronine)

L12 ANSWER 29 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1993:625943 HCAPLUS
DOCUMENT NUMBER: 119:225943
TITLE: Antidiabetic thiazolidine compounds
INVENTOR(S): Yoshioka, Takao; Nishi, Takahide; Kanai, Tsutomu; Aizawa, Yuichi; Wada, Kunio; Fujita, Takashi; Horikoshi, Hiroyoshi
PATENT ASSIGNEE(S): Sankyo Co., Ltd., Japan
SOURCE: Eur. Pat. Appl., 58 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| EP 549365 | A1 | 19930630 | EP 1992-311813 | 19921224 |
| EP 549365 | B1 | 19950809 | | |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE | | | | |
| NO 9204964 | A | 19930628 | NO 1992-4964 | 19921222 |
| ZA 9210021 | A | 19930702 | ZA 1992-10021 | 19921223 |
| HU 67788 | A2 | 19950428 | HU 1992-4130 | 19921223 |

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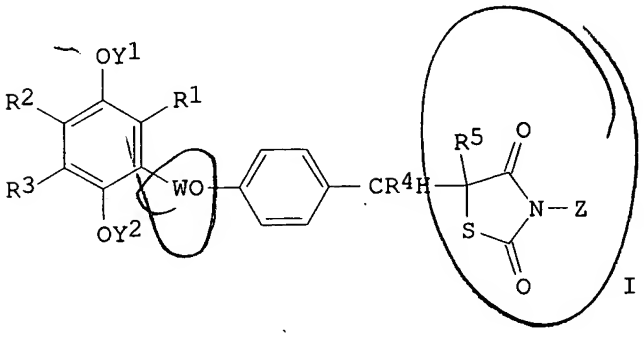
|---|---|---|---|---|---|
| HU 215450 | B | 19990428 | | | |
| CA 2086277 | AA | 19930627 | CA 1992-2086277 | 19921224 | |
| AU 9230431 | A1 | 19930701 | AU 1992-30431 | 19921224 | |
| AU 654223 | B2 | 19941027 | | | |
| ES 2078671 | T3 | 19951216 | ES 1992-311813 | 19921224 | |
| JP 05239041 | A2 | 19930917 | JP 1992-346122 | 19921225 | |
| JP 2833949 | B2 | 19981209 | | | |
| IL 104238 | A1 | 19961031 | IL 1992-104238 | 19921225 | |
| RU 2095354 | C1 | 19971110 | RU 1992-16231 | 19921225 | |
| CN 1074680 | A | 19930728 | CN 1992-115234 | 19921226 | |
| CN 1032751 | B | 19960911 | | | |

PRIORITY APPLN. INFO.: JP 1991-344571 A 19911226
OTHER SOURCE(S): MARPAT 119:225943
GI




AB The title compds. I [R1 = C1-5 alkyl; R2, R3 = C1-5 alkyl, C1-5 alkoxy; R4, R5 = H; Y1, Y2 = H, C1-5 alkyl, C1-7 aliph. carboxylic acyl group, benzoyl, naphthoyl, pyridinecarbonyl, (un)substituted quinolinecarbonyl; W = direct bond, C1-5 alkylene group; Z = H, cation; R2R3 = (un)substituted benzene ring and R1 = H, halogen, alkyl; R4R5 = single double C-C bond], useful in the treatment of adult-onset diabetes or hyperlipidemia (no data), are prepd. Thus, 5-[4-(2,5-dihydroxy-3,4,6-trimethylphenoxy)benzyl]thiazolidine-2,4-dione was esterified in the presence of Ac2O, producing 5-[[4-(2,5-diacetoxy-3,4,6-trimethylphenoxy)benzyl]thiazolidine-2,4-dione (II). II demonstrated blood glucose lowering rate [[(blood glucose level of rat group given placebo - blood glucose level in rat group administered test compds.)/blood glucose level in rat group administered placebo] x 100] of 24.0%.

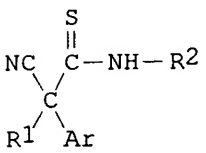
IT **150556-02-8P**
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. and reaction. of, in prepn. of **antidiabetic** agents)

L12 ANSWER 30 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1993:559902 HCAPLUS
DOCUMENT NUMBER: 119:159902
TITLE: Preparation of cyanophenylthioacetamides as antihypertensives.
INVENTOR(S): Okujima, Hiromi; Niifuku, Tetsuo; Betsusho, Hideki; Kyono, Asami; Hayashi, Junko; Tobe, Akihiro; Kobayashi, Makio

PATENT ASSIGNEE(S): Mitsubishi Chem Ind, Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 18 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| JP 05059003 | A2 | 19930309 | JP 1991-224411 | 19910904 |

OTHER SOURCE(S): MARPAT 119:159902
GI



I

AB The title compds. [I; R1 = H, alkyl, cycloalkyl, (CH2)n-A; A = (un)substituted aryl, heterocyclyl; R2 = alkyl; n = 0, 1-6 integer; Ar = aryl, heterocyclyl] are prepd. E.g., KOCMe3 was added to a mixt. of 4-(1H-imidazol-1-yl)acetophenone, EtOH, and p-toluenesulfonylmethyl isocyanide in MeOCH2CH2OMe and the resulting mixt. was stirred at room temp. for 0.5 h and then warmed at 40.degree. for 2 h to give 52% 2-[4-(1H-imidazol-1-yl)phenyl]propionitrile, which was treated with KOCMe3 and MeNCS in THF at room temp. for 2 h to give I [R1 = H, R2 = Me, Ar = 4-(1H-imidazol-1-yl)phenyl]. This at 100 mg/Kg p.o. reduced the blood pressure of spontaneously hypertensive rats by 97.8 mmHg.

IT **137274-86-3P**
RL: BAC (Biological activity or effector, except adverse); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. of, as **antihypertensive**)

L12 ANSWER 31 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1992:34484 HCAPLUS
DOCUMENT NUMBER: 116:34484
TITLE: Triglyceride and cholesterol concentrations in serum during chronic lithium therapy: a retrospective comparison of baseline and follow-up laboratory values
AUTHOR(S): Fankhauser, M.; Krueger, R.; Finley, P.
CORPORATE SOURCE: Coll. Pharm., Univ. Arizona, Tucson, AZ, 85721, USA
SOURCE: Lithium (1991), 2(2), 77-81
CODEN: LITHER; ISSN: 0954-1381
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A retrospective chart review of outpatient medical records was done at a community mental health center to det. whether changes occurred in baseline serum triglyceride and cholesterol concns. during chronic lithium

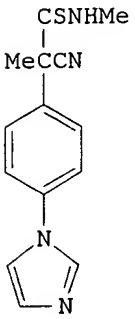
administration. The authors compared the serum triglyceride and cholesterol concns. at baseline (pre-lithium) with those obtained after lithium therapy in 24 patients. The serum triglyceride concns. measured after 12 mo on lithium therapy were higher than baseline concns. Despite the increase in triglyceride levels during lithium therapy, there was no change in serum cholesterol concns. A comparison of thyroid function tests indicated a redn. of T3U after 49 mo of lithium therapy in comparison to baseline concns. Further controlled studies are needed to det. whether changes in serum triglyceride concns. during chronic lithium therapy are related to wt. gain, diet, alterations in thyroid functioning, concomitant medications or the disease state.

IT **6893-02-3**, Triiodothyronine
RL: BIOL (Biological study)
(in blood serum, of human, chronic lithium **therapy** effect on **triglyceride** and cholesterol and)

L12 ANSWER 32 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1991:679613 HCAPLUS
DOCUMENT NUMBER: 115:279613
TITLE: Preparation of .alpha.-thiocarbamoyl-.alpha.-arylacetonitriles as antihypertensives
INVENTOR(S): Okushima, Hiromi; Tobe, Akihiro; Kobayashi, Makio; Shimpuku, Tetsuro; Bessho, Hideki; Hayashi, Junko; Seino, Asami
PATENT ASSIGNEE(S): Mitsubishi Kasei Corp., Japan
SOURCE: Eur. Pat. Appl., 30 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| EP 445698 | A1 | 19910911 | EP 1991-103220 | 19910304 |
| EP 445698 | B1 | 19940622 | | |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE | | | | |
| JP 04211048 | A2 | 19920803 | JP 1991-34614 | 19910228 |
| CA 2037483 | AA | 19910906 | CA 1991-2037483 | 19910304 |
| US 5246958 | A | 19930921 | US 1991-664053 | 19910304 |
| ES 2055476 | T3 | 19940816 | ES 1991-103220 | 19910304 |

PRIORITY APPLN. INFO.: JP 1990-53309 19900305
OTHER SOURCE(S): MARPAT 115:279613
GI



II

AB R1C(CN)(Ar)CSNHR2 [I; R1 = H, C1-6 alkyl, C3-6 cycloalkyl, (CH2)nR; R = C6-12 aryl, (substituted) 5- or 6-membered (fused) heterocyclyl, e.g., imidazolyl; n = 0-6; R2 = C1-10 alkyl; Ar = (substituted) aryl, (substituted) 5- or 6-membered (fused) heterocyclyl] were prepd. as antihypertensives. Thus, Me3COK was added with cooling to a soln. of 4-(1-imidazolyl)acetophenone and 4-MeC6H4SO2NC in ethylene glycol and the mixt. was stirred at room temp. for 0.5 h, then stirred at 40.degree. for 2 h. The propionitrile formed was dissolved in THF and condensed with MeNCS in the presence of Me3COK to give title compd. II. II at 10 mg/kg decreased av. blood pressure by 97.8 mmHg for hypertensive rats with av. initial blood pressure of 173.4 mmHg after 2 h.

IT **137274-86-3P**
RL: BAC (Biological activity or effector, except adverse); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. of, as **antihypertensive**)

L12 ANSWER 33 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1989:433486 HCAPLUS
DOCUMENT NUMBER: 111:33486
TITLE: Helpless behavior (escape deficits) in streptozotocin-diabetic rats: resistance to antidepressant drugs
AUTHOR(S): Massol, Jacques; Martin, Patrick; Belon, Jean Paul; Puech, Alain J.; Soubrie, Philippe
CORPORATE SOURCE: Dep. Pharmacol., Fac. Med. Pitie-Salpetriere, Paris, 75634, Fr.
SOURCE: Psychoneuroendocrinology (Oxford) (1989), 14(1-2), 145-53
CODEN: PSYCDE; ISSN: 0306-4530
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The possibility of an impaired response to antidepressant drugs in diabetic rats was studied using the learned helplessness model of depression. Exptl. diabetes was induced by 3 i.p. injections of streptozotocin (3795, 37.5, 50 mg/kg, 3 days apart), four weeks before behavioral testing. Diabetic and non-diabetic rats were first exposed to 60 inescapable shocks. Twice daily (i.p.) injection of clomiprasmine (24 mg/kg), desipramine (24 mg/kg), imipramine (32 mg/kg) or clenbuterol (0.75

mg/kg) prevented escape deficits in the non-diabetic but not in the diabetic rats. Moreover, one week of insulin therapy restored operant escape responding to both the tricyclics and a .beta.-agonist. The inefficacy of clenbuterol (a central .beta.-agonist) in reversing helpless behavior in diabetic rats, along with the observation that triiodothyronine (T3) supplementation also restored the response to imipramine in the diabetic rats, suggests that thyroid-mediated alterations of central noradrenergic function might be a crit. factor in the resistance or delayed response to antidepressants in exptl. diabetes.

IT **6893-02-3**, Triiodothyronine
RL: BIOL (Biological study)
(resistance to antidepressant **drugs** in **diabetes** response to)

L12 ANSWER 34 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1989:166077 HCAPLUS
DOCUMENT NUMBER: 110:166077
TITLE: Impaired response of experimental diabetic mice to tricyclics: a possible beta-adrenergic mechanism
AUTHOR(S): Massol, Jacques; Martin, Patrick; Chatelain, Francoise; Soubrie, Philippe; Puech, Alain Jacques
CORPORATE SOURCE: Serv. Diabetol.-Endocrinol., Hop. J. Minjoz, Besancon, 25030, Fr.
SOURCE: Pharmacol., Biochem. Behav. (1988), 31(4), 807-12
CODEN: PBBHAU; ISSN: 0091-3057
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Diabetes is reportedly assocd. with alterations in peripheral and central noradrenergic systems. The latter might be involved in the antidepressant effects of imipramine-like drugs in both humans and animals. Therefore, it is possible that diabetics show an impaired responsiveness to tricyclics. To test this possibility the effects of streptozotocin (STZ)-induced exptl. diabetes in mice were assessed in two psychopharmacol. tests: (1) the reversal of apomorphine (16 mg/kg)-induced hypothermia and (2) the hypoactivity induced by a direct .beta.-agonist (clenbuterol 0.06 mg/kg). At day 15 after STZ or vehicle treatment, imipramine (4 mg/kg) antagonized the apomorphine-induced hypothermia in diabetic (D) and nondiabetic (ND) mice and clenbuterol produced hypoactivity in both groups. At day 30 and 45, the ability of imipramine (1, 2, 4, 8, 16 mg/kg), clomipramine (8 mg/kg) and desipramine (2 mg/kg) to reverse apomorphine-induced hypothermia disappeared at the same time that clenbuterol lost its ability to induce hypomotility in D mice. These impaired responses on both tests were cor. by a short period of insulin therapy. These two tests may reflect central .beta.-adrenergic functions. Therefore, these data suggest that the impaired responsiveness of diabetic mice might be due at least in part to a noradrenergic dysfunction. Possibly, in diabetes, a .beta.-adrenoceptor desensitization identical to that obsd. at the peripheral level occurs in the central nervous system. The possibility that a thyroid hormone deficiency may be involved was also tested. Decreased T3 plasma levels were found in D mice concomitant with the impaired pharmacol. responses and T3 supplementation returned these responses to normal. An hypothetical link between these thyroid effects and the .beta.-adrenergic desensitization in D mice could be suggested but remains to be detd.

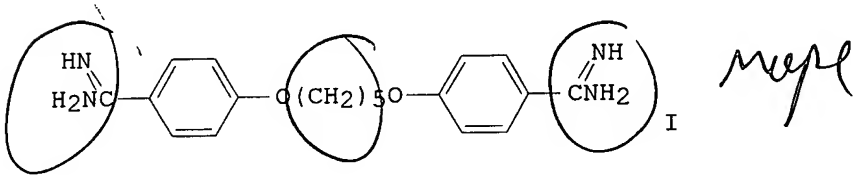
IT **6893-02-3**, Triiodothyronine
RL: BIOL (Biological study)
(antidepressants **pharmacol.** in **diabetes** response to)

L12 ANSWER 35 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1985:589750 HCAPLUS
DOCUMENT NUMBER: 103:189750
TITLE: Hormone-induced changes in response to drugs affecting cardiac function and metabolism
AUTHOR(S): Hess, Marilyn E.
CORPORATE SOURCE: Sch. Med., Univ. Pennsylvania, Philadelphia, PA, 19104, USA
SOURCE: Dev. Cardiovasc. Med. (1985), 46(Pathog. Stress-Induced Heart Dis.), 172-84
CODEN: DCMEDM; ISSN: 0166-9842
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Supersensitivity of the metabolic effects of catecholamines in the heart in hyperthyroidism and **diabetes** and its modulation by **drugs**, diet, or altered hormonal states is discussed with respect to the role of myocardial phosphorylase [9035-74-9]. Desmethylimipramine [50-47-5] prevented the enhanced stimulation of phosphorylase a by isoproterenol [7683-59-2] in rat heart after treatment with thyroxine [**51-48-9**]; similar results were noted in **diabetic** rats. Desmethylimipramine prevented the increase in the no. of rat cardiac .beta.-adrenergic receptors caused by thyroxine; however, no effect of the **drug** was apparent on the decrease in the [3H]quinuclidinyl benzilate binding to heart muscarinic receptors. Results from expts. on the modulation of cardiac glycogen [9005-79-2] content and uridine kinase [9026-39-5] activity in normal and **diabetic** rats by high-carbohydrate and high-carbohydrate diets contg. acarbose [56180-94-0] are presented.

L12 ANSWER 36 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1985:179591 HCAPLUS
DOCUMENT NUMBER: 102:179591
TITLE: Endocrine control of thymic serum factor production in young-adult and old mice
AUTHOR(S): Fabris, N.; Mocchegiani, E.
CORPORATE SOURCE: Res. Dep., INRCA, Ancona, 60100, Italy
SOURCE: Cell. Immunol. (1985), 91(2), 325-35
CODEN: CLIMB8; ISSN: 0008-8749
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The influence of different endocrinol. manipulations on the blood concn. of serum thymic factor (FTS) [78922-62-0] was studied in young-adult and old mice. Among the exptl. induced endocrinopathies in youth, hypothyroidism and **diabetes** caused strong redns. of FTS levels, which were restored to normal by the appropriate hormonal substitutive **therapy**. Removal of adrenals or gonads has no effect on FTS level. Old mice, which show undetectable levels of FTS and low levels of thyroxine [**51-48-9**], regain the capacity to produce FTS, provided they are treated with thyroxine. The variations of FTS blood

levels in the course of endocrinol. manipulations were due to a direct or indirect effect exerted on the recipient thymus. Hormonal treatment of thymectomized mice did not induce any FTS-like activity in their sera, nor did hormones interfere in vitro with the bioassay used to test for FTS. Apparently, the neuroendocrine balance modulates the synthesis and(or) the release of FTS from the thymus during the whole life of the organism and decline of FTS prodn. with advancing age is largely dependent on age-assocd. endocrinol. imbalances.

L12 ANSWER 37 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1985:346 HCAPLUS
DOCUMENT NUMBER: 102:346
TITLE: Hypotensive effect of aromatic amidines and imidazolines
AUTHOR(S): Bielenberg, G. W.; Krieglstein, J.
CORPORATE SOURCE: Inst. Pharmakol. Toxikol., Philipps-Univ., Marburg/Lahn, 3550, Fed. Rep. Ger.
SOURCE: Arzneim.-Forsch. (1984), 34(9), 958-67
CODEN: ARZNAD; ISSN: 0004-4172
DOCUMENT TYPE: Journal
LANGUAGE: German
GI



I

AB Of 16 arom. amidines or imidazolines tested for hypotensive activity in exptl. animals, all substances, except for 5-amidino-2-phenylindole (271/179) [93490-77-8], caused a dose-dependent hypotensive effect. Pentamidine (I) [100-33-4] was one of the most effective hypotensives. The biscationic character of a compd. was a prerequisite for strong antihypertensive activity. The antihypertensive activity of the most active compds. appeared to have a peripheral origin and did not appear to be mediated via parasympathomimetic or histaminic mechanisms. Cardiovascular effects of these compds. are also given. The antihypertensive activity of these compds. is discussed in terms of a musculotropic action on vascular smooth muscle.

IT **73819-47-3 73819-49-5**
RL: BAC (Biological activity or effector, except adverse); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**antihypertensive** activity of)

L12 ANSWER 38 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1984:545790 HCAPLUS
DOCUMENT NUMBER: 101:145790
TITLE: The 8-hour metabolic profile after drinking ethanol
AUTHOR(S): Joffe, B. I.; Kalk, W. J.; Shires, R.; Lamprey, J. M.; Baker, S.; Seftel, H. C.
CORPORATE SOURCE: Med. Sch., Univ. Witwatersrand, Johannesburg, S. Afr.

SOURCE: J. Endocrinol. Invest. (1984), 7(3), 239-41
CODEN: JEIND7; ISSN: 0391-4097
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The acute metabolic changes after drinking EtOH [64-17-5] were studied in 11 fasting, healthy, nonobese, **medical** students, 6 of whom consumed 40 g EtOH dild. with 750 mL of a sugar-free soft drink over 1 h. The other 5 drank the same vol. of soft drink alone. **Blood** levels of EtOH, **glucose**, immunoreactive insulin [9004-10-8], and growth hormone [9002-72-6] were measured over the ensuing 8 h, as well as the plasma concns. of prolactin [9002-62-4], cortisol [50-23-7], and triiodothyronine [**6893-02-3**]. After ingesting EtOH, the mean plasma glucose concn. declined, but not to hypoglycemic levels (the nadir was 3.9 mmol/L at 6 h), insulin levels fell gradually, and the mean growth hormone concn. showed a modest late rise. Other hormones did not change significantly. Thus, in the particular setting examd., the oral administration of EtOH does not cause hypoglycemia or other adverse effects on carbohydrate metab.

L12 ANSWER 39 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1984:523835 HCAPLUS
DOCUMENT NUMBER: 101:123835
TITLE: Effects of hormones, fasting and diabetes on triglyceride lipase activities in rat heart and liver
AUTHOR(S): Stam, H.; Schoonderwoerd, K.; Breeman, W.; Huelsmann, W. C.
CORPORATE SOURCE: Med. Fac., Erasmus Univ., Rotterdam, 3000 DR, Neth.
SOURCE: Horm. Metab. Res. (1984), 16(6), 293-7
CODEN: HMMRA2; ISSN: 0018-5043
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effects of Kenacort [124-94-7], Synacthen [16960-16-0], L-thyroxine [**51-48-9**], fasting, and exptl. **diabetes** on the activities of acid, neutral, and alk. **triglyceride** lipase [9001-62-1] activities in the heart and liver of rats were studied. Cardiac lipoprotein lipase (EC 3.1.1.34) [9004-02-8] activity was increased after fasting, exptl. **diabetes**, and all 3 hormone treatments. Cardiac neutral lipase activity was decreased during **diabetes** and was enhanced during fasting and by the hormone treatments. Myocardial acid lipase activity was decreased during fasting and corticosteroid administration but was not affected by the short-term ACTH treatment. Hepatic acid lipase activity was increased during fasting, **diabetes**, and thyroxine treatment but was decreased by ACTH and corticosteroid **therapy**. The liver alk. phosphatase [9001-78-9] activity was depressed by fasting, **diabetes**, corticosteroid, and ACTH and was slightly increased by thyroxine. The possible mechanism underlying the obsd. changes in acid, neutral, alk., and lipoprotein lipase activities in the heart and liver were discussed.
IT **51-48-9**, biological studies
RL: BIOL (Biological study)
(**triglyceride** lipase of heart and liver response to)

L12 ANSWER 40 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1984:466575 HCAPLUS

DOCUMENT NUMBER: 101:66575
TITLE: Lack of effect of thyroid hormone on diabetic rat heart function and biochemistry
AUTHOR(S): Tahiliani, Arun G.; McNeill, John H.
CORPORATE SOURCE: Fac. Pharm. Sci., Univ. British Columbia, Vancouver, BC, V6T 1W5, Can.
SOURCE: Can. J. Physiol. Pharmacol. (1984), 62(6), 617-21
CODEN: CJPPA3; ISSN: 0008-4212
DOCUMENT TYPE: Journal
LANGUAGE: English

AB To study the degree of involvement of **diabetes**-induced hypothyroidism on altered myocardial function, thyroid replacement **therapy** was carried out in streptozotocin-**diabetic** rats. T3 [**6893-02-3**] treatment was initiated 3 days after the rats were made **diabetic** and was carried out for 6 wk thereafter. Isolated perfused hearts from **diabetic** rats exhibited a depression in left ventricular developed pressure and pos. and neg. dP/dt at higher filling pressures as compared with controls. The depression was not prevented by thyroid treatment. Ca uptake activity in the cardiac sarcoplasmic reticulum (SR) was also depressed as a result of **diabetes** and this depression also was not prevented by thyroid treatment. Long-chain acyl carnitine levels were elevated in **diabetic** cardiac SR and were not lowered by T3 treatment. The myocardial dysfunction obsd. in **diabetic** rats is apparently due to factors other than the induced hypothyroidism.

IT **6893-02-3**
RL: BIOL (Biological study)
(heart function response to, in **diabetes**)

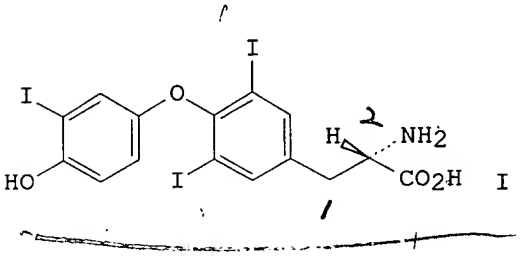
L12 ANSWER 41 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1984:401326 HCAPLUS
DOCUMENT NUMBER: 101:1326
TITLE: Effect of oral hypoglycemic drugs, triiodothyronine and their interaction on nucleotide metabolism in maturity onset diabetics
AUTHOR(S): Hafiez, A. A.; Ismail, A. A.; El-Kirdassy, Z. H.; Sharada, H. M.
CORPORATE SOURCE: Fac. Med., Cairo Univ., Cairo, Egypt
SOURCE: Isotopenpraxis (1984), 20(5), 193-8
CODEN: IPRXA9; ISSN: 0021-1915
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Blood cAMP [60-92-4] levels were lower in **diabetic** patients than in controls, but other adenine nucleotides did not vary. CAMP levels of **diabetics** were restored to normal by treatment for 5 days with triiodothyrosine [**6893-02-3**] or with the oral **antidiabetic drugs** glibenclamide [10238-21-8] or gliclazide [21187-98-4]. Combined treatment with triiodothyronine and gliclazide caused an even greater increase in cAmP levels, and it also increased ATP [56-65-5]. Triiodothyronine raised cAMP levels in **diabetes** without affecting **blood sugar**, whereas both **antidiabetic drugs** increased cAMP and decreased **blood sugar**. Basal plasma cAMP levels were lower in women than in men, both in normals and in **diabetics**.

IT **6893-02-3**
RL: BIOL (Biological study)
(adenine nucleotides of blood in response to, in **diabetic** humans)

L12 ANSWER 42 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1983:469340 HCAPLUS
DOCUMENT NUMBER: 99:69340
TITLE: Experimental substantiation of the methods for increasing the efficiency of parenteral nutrition
AUTHOR(S): Skovronskaya, E. V.; Vovk, G. P.
CORPORATE SOURCE: L'vov. Nauchno-Issled. Inst. Gematol. Pereliv. Krovi, Lvov, USSR
SOURCE: Gematol. Transfuziol. (1983), 28(5), 58-61
CODEN: GETRE8
DOCUMENT TYPE: Journal
LANGUAGE: Russian
AB Using rat model expts., parenteral feeding of casein hydrolyzates, polyamines, amino acids (methionine, lysine, tryptophan), glucose, Intralipid, vitamins, insulin [9004-10-8], nerobolil [62-90-8], and kontrikal (proteinase inhibitor) [9075-10-9], in various combinations, was used as **therapeutic** treatment for protein deficiency, alloxan **diabetes**, hepatitis and thyroxine [**51-48-9**] intoxication. Emphasis was placed on parenteral feeding in maintenance of N balance.
IT **51-48-9**, biological studies
RL: BIOL (Biological study)
(intoxication from, parenteral feeding as **therapy** for)

L12 ANSWER 43 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1983:448222 HCAPLUS
DOCUMENT NUMBER: 99:48222
TITLE: Thyroid hormone and lipoprotein metabolism
AUTHOR(S): Mabuchi, Hiroshi
CORPORATE SOURCE: Med. Sch., Kanazawa Univ., Kanazawa, Japan
SOURCE: Domyaku Koka (1982), 10(4), 605-9
CODEN: DOMKDM; ISSN: 0386-2682
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
GI



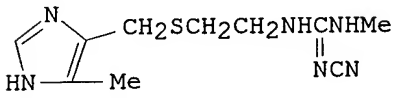

AB Treatment of 8 humans with hypothyroidism with thyroid hormone resulted in

decreased levels of serum cholesterol [57-88-5] and **triglycerides** and increased levels of serum lipoprotein lipase (LPL) [9004-02-8] and hepatic **triglyceride** lipase (HTGL) [9001-62-1]. Treatment of 5 cases of hyperthyroidism with thyroid hormone resulted in a slightly increased level of LPL has a significantly decreased level of serum HTGL. Patients with hypothyroidism showed a decreased level of serum lecithin-cholesterol acyltransferase (LCAT) [9031-14-5], whereas those with hyperthyroidism showed an increased level of serum LCAT. Treatment with thyroid hormone tended to restore the enzyme concns. to normal values. Blood thyroid hormone levels correlated pos. with the blood LCAT activity. In a case of hyperthyroidism complicated by familial hypercholesterolemia, thyroid hormone **therapy** elevated the low-d. lipoprotein (LDL) receptor activity and lowered the blood cholesterol level. In an in vitro expt. with cultured human fibroblasts, T3 (I) [**6893-02-3**] (1.0 .mu.g/mL) caused a 11-29% increase in LDL receptor activity. In the liver isolated from thyroidectomized rats, I supplement increased the incorporation of [14C]HOAc into cholesterol to above normal levels. This was accompanied by an increase in hydroxymethylglutaryl CoA reductase [9028-35-7] activity. These findings are discussed with respect to the effect of thyroid hormone on lipoprotein metab.

L12 ANSWER 44 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1983:155444 HCAPLUS
DOCUMENT NUMBER: 98:155444
TITLE: Cardiac function and myosin ATPase in diabetic rats treated with insulin, T3, and T4
AUTHOR(S): Garber, David W.; Everett, Alan W.; Neely, James R.
CORPORATE SOURCE: Milton S. Hershey Med. Cent., Pennsylvania State Univ., Hershey, PA, 17033, USA
SOURCE: Am. J. Physiol. (1983), 244(4), H592-H598
CODEN: AJPHAP; ISSN: 0002-9513
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effects of insulin [9004-10-8], T4 [**51-48-9**] and T3 [**6893-02-3**] treatment on cardiac function, myosin ATPase [9000-83-3] activity, and myosin isozyme distribution were studied in alloxan-**diabetic** rats. **Diabetes** depressed peak ventricular pressure development, heart rate, and the max. rate of left ventricular pressure development. Myocardial Ca2+-activated myosin ATPase activity was reduced in assocn. with lower serum levels of T3 and T4. The V1 isozyme of myosin decreased, and both V2 and V3 isozymes increased. Insulin treatment totally reversed the changes in function, serum thyroid hormones, and myosin ATPase activity. Treatment of **diabetic** animals with T4 (5 or 10 .mu.g/day) prevented the decrease in myosin ATPase but did not prevent the changes in cardiac function, myosin isozymes, or serum T3 levels. **Pharmacol.** doses of T3 (3 .mu.g/day) that were adequate to maintain higher than normal serum T3 cor. the decrease in Ca2+-activated myosin ATPase and heart rate but only partially cor. the changes in pressure development and myosin isozyme distribution. Only when serum T3 was increased to 4 times normal was cardiac function cor.
IT **51-48-9**, biological studies **6893-02-3**
RL: BIOL (Biological study)

(heart function and myosin ATPase and isozymes response to, in **diabetes**)

L12 ANSWER 45 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1983:119449 HCAPLUS
DOCUMENT NUMBER: 98:119449
TITLE: The endocrine and metabolic effects of cimetidine
AUTHOR(S): Stubbs, W. A.; Delitala, G.; Besser, G. M.; Edwards, C. R. W.; Labrooy, S.; Taylor, R.; Misiewicz, J. J.; Alberti, K. G. M. M.
CORPORATE SOURCE: Dep. Med. Endocrinol., St. Bartholomew's Hosp., London, EC1A 7BE, UK
SOURCE: Clin. Endocrinol. (Oxford) (1983), 18(2), 167-78
CODEN: CLECAP; ISSN: 0300-0664
DOCUMENT TYPE: Journal
LANGUAGE: English
GI

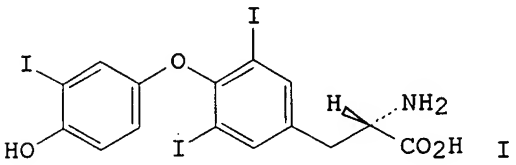


I

AB Serial blood sampling in patients treated with cimetidine (I) [51481-61-9] (1 g/day) showed that prolactin (PRL) [9002-62-4] values were within the normal range apart from a stress-induced initial rise. Hormonal and metabolic profiles from 08:30 to 18:30 h were performed in patients before and after 1 mo treatment with cimetidine (1 g/day). Circulating PRL, LH [9002-67-9], FSH [9002-68-0], growth hormone [9002-72-6], TSH [9002-71-5], T3 [**6893-02-3**], T4 [**51-48-9**], and testosterone [58-22-0] were similar before and after treatment. The mean **blood glucose** fell from 5.4 to 4.8 mM in patients on cimetidine. Small changes were also obsd. in blood pyruvate [127-17-3], lactate [50-21-5], 3-hydroxybutyrate [300-85-6] and the lactate/pyruvate ratio. The effects of oral or i.v. cimetidine on the circulating concns. of insulin [9004-10-8], glucose and intermediary metabolites were investigated in normal subjects. I.v. cimetidine (100 mg/h for 4 h) given to fasting subjects decreased **blood glucose** and serum insulin by 15 and 34%, resp., at 150 min. During an oral glucose tolerance test (GTT), i.v. cimetidine caused a striking decline in **blood glucose**, lactate, and pyruvate responses compared with control studies, although the serum insulin was similar to control values. When given for 48 h before the study, oral cimetidine did not alter basal serum insulin and **blood glucose**, lactate, pyruvate, alanine [56-41-7], glycerol [56-81-5] and 3-hydroxybutyrate levels. However, 150 min after an oral GTT the serum insulin was increased by 47% by oral cimetidine although the **blood glucose** was not significantly changed compared with the control day. Oral cimetidine had no effect on the **blood glucose** or serum insulin during an i.v. GTT. Apparently, oral cimetidine given at **therapeutic** doses to patients with peptic ulcers does not produce consistent changes in circulating anterior

pituitary hormones. Oral cimetidine given to patients for 1 mo and i.v. cimetidine given to normal subjects have mild hypoglycemic effects. Oral cimetidine administered over 48 h to normal subjects has little effect on **blood glucose** concn.

L12 ANSWER 46 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1982:136481 HCAPLUS
DOCUMENT NUMBER: 96:136481
TITLE: Influence of thyroid hormone administration on myosin ATPase activity and myosin isoenzyme distribution in the heart of diabetic rats
AUTHOR(S): Dillmann, Wolfgang H.
CORPORATE SOURCE: Dep. Med., Univ. California, San Diego, CA, 92103, USA
SOURCE: Metab., Clin. Exp. (1982), 31(3), 199-204
CODEN: METAAJ; ISSN: 0026-0495
DOCUMENT TYPE: Journal
LANGUAGE: English
GI

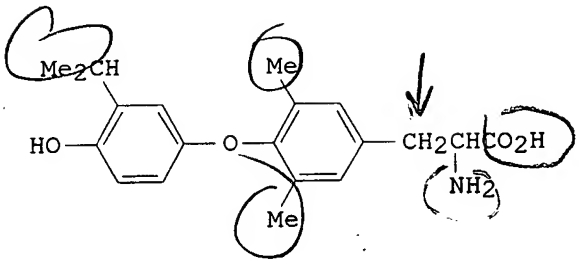


AB Streptozotocin-induced **diabetes** in rats lowered the myosin ATPase [9000-83-3] activity and altered the myosin isoenzyme distribution, and injections with physiol. replacement doses of triiodothyronine (I) [**6893-02-3**] (0.3 .mu.g/100 g/day for 4 wk) did not restore either the enzyme activity or isoenzyme distribution. However, injections of **pharmacol**. doses of I or thyroxine (II) [**51-48-9**] (3 .mu.g I or 10 .mu.g II/100 g/day for 4 wk) normalized both parameters. The lack of response to physiol. replacement doses may indicate a decreased responsiveness to thyroid hormones in **diabetic** animals, or myosin formation may be influenced by factors other than thyroid hormones.

IT **51-48-9**, biological studies **6893-02-3**
RL: BIOL (Biological study)
(myosin ATPase of heart response to, in **diabetes**)

L12 ANSWER 47 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1982:98135 HCAPLUS
DOCUMENT NUMBER: 96:98135
TITLE: 3,5-Dimethyl-3'-isopropyl-L-thyronine therapy in diabetic pregnancy. Stimulation of rabbit fetal lung phospholipids
AUTHOR(S): Neufeld, Naomi; Melmed, Shlomo
CORPORATE SOURCE: Sch. Med., Univ. California, Los Angeles, CA, 90048, USA
SOURCE: J. Clin. Invest. (1981), 68(6), 1605-9

CODEN: JCINAO; ISSN: 0021-9738
DOCUMENT TYPE: Journal
LANGUAGE: English
GI




I

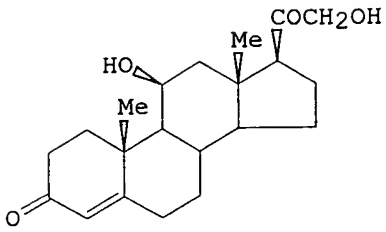
no allyl groups

AB **Diabetes** mellitus in pregnancy is assocd. with neonatal respiratory distress syndrome due to impaired synthesis of fetal lung surfactant. Maternal administration of 3,5-dimethyl-3'-isopropyl-L-thyronine (I) [**26384-44-1**] enhanced fetal lung phospholipid synthesis and accelerated lung maturity. Therefore the effects of I (0.5 mg/kg/day, s.c.) administered to pregnant alloxan-**diabetic** rabbits on days 25 and 26 of gestation were examd. I treatment of **diabetic** maternal rabbits (DD) decreased maternal **blood glucose** (115 vs. 275 mg/dL) and fetal glucose (64 vs. 274 mg/dL) compared with saline-injected **diabetic** (D) mothers. A decrease of fetal insulin [9004-10-8] levels was also assocd. with maternal I **therapy** in **diabetic** rabbits. Maternal **diabetes** also decreased fetal lung wt. (370 vs. 520 mg) and lung protein content (6.5 vs. 8.7 mg/gm), both of which were restored to normal in offspring of I-treated **diabetic** rabbits. Maternal I administration decreased fetal lung glycogen [9005-79-2] content in control (62 vs. 25 .mu.g/mg protein) and **diabetic** (56 vs. 34 .mu.g/mg protein) offspring. Whereas maternal **diabetes** was assocd. with decreases of all major phospholipid species in fetal lung-comprising surfactant, these were restored with I **therapy**. Thus, short-term maternal administration of I in pregnant **diabetic** rabbits not only promotes fetal lung phospholipid synthesis, but also appears to ameliorate maternal hyperglycemia. Thus, I is of potential benefit in the management of **diabetic** pregnancy.

IT **26384-44-1**
RL: BIOL (Biological study)
(phospholipid formation by embryo lung stimulation by, in **diabetic** pregnancy)

L12 ANSWER 48 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1981:435886 HCAPLUS
DOCUMENT NUMBER: 95:35886
TITLE: Capacity of corticosterone receptor system in rat brain: control by neuropeptides and hormones
AUTHOR(S): Veldhuis, Dick; De Kloet, Ronald
CORPORATE SOURCE: Med. Fac., Univ. Utrecht, Utrecht, 3521 GD, Neth.
SOURCE: Adv. Physiol. Sci., Proc. Int. Congr., 28th (1981), Meeting Date 1980, Volume 13, Issue Endocrinol., Neuroendocrino., Neuropept., Pt. 1, 61-5. Editor(s):

Stark, E.; Makara, G. B.; Acs, Zs. Akad. Kiado: Budapest, Hung.
CODEN: 45TGAW
DOCUMENT TYPE: Conference
LANGUAGE: English
GI




I

AB In rats, hippocampal corticosterone (I) [50-22-6] receptor capacity increases after removal of the pituitary and is decreased in rats homozygous for **diabetes** insipidus. Replacement **therapy** with various hormones and neuropeptides showed that the homologous hormone, thyroxine [**51-48-9**] and testosterone [58-22-0] as well as peptides related to arginine-vasopressin [113-79-1] and ACTH all are involved in the control of hippocampal I receptor capacity.

L12 ANSWER 49 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1980:419560 HCAPLUS
DOCUMENT NUMBER: 93:19560
TITLE: Hormonal regulation of liver pyruvate kinase concentration and activity
AUTHOR(S): Johnson, Mark L.; Veneziale, Carlo M.
CORPORATE SOURCE: Sect. Biochem., Mayo Med. Sch., Rochester, MN, USA
SOURCE: Biochemistry (1980), 19(10), 2191-5
CODEN: BICHAW; ISSN: 0006-2960
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The hormonal control of rabbit liver (L type) pyruvate kinase (EC 2.7.1.40) [9001-59-6] concn. and activity was investigated. The liver maintained the enzyme concn. (nanomoles per g of liver) within narrow limits. The concn. decreased slightly after fasting and the administration of glucagon [9007-92-5] or triamcinolone [124-94-7]. The total organ amt. (nanomoles) of enzyme changed but mainly because of changes in liver wt. Liver pyruvate kinase from rabbits fed a control diet which was 50-60% carbonhydrate had a specific activity of 12.7 units/nmol of enzyme. Starvation and glucagon each lowered the specific activity to 10.2 units/nmol. Alloxan **diabetes** also resulted in a decrease which could be reversed by insulin [9004-10-8] **therapy** . Triamcinolone decreased the enzyme specific activity to 7.4 units/umol. Thyroxine [**51-48-9**] caused the enzyme to have a slightly higher value of 14.0 units/nmol. These changes in specific activity resulted mainly from altered enzyme activity (units per g of liver). Total organ activity was reduced in fasting and after triamcinolone administration.

Primarily this was due to a decrease in liver wt. and enzyme activity (units per g of liver) in fasting and to a decrease in enzyme activity after triamcinolone administration. In both states a decrease in enzyme concn. also contributed. Thus, regulation of liver pyruvate kinase is mainly through regulation of the specific activity, i.e. the catalytic state, of the enzyme. However, in the assessment of total hepatic glycolysis and gluconeogenesis, changes in total organ enzyme activity based on changes in organ wt. must also be considered.

L12 ANSWER 50 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1980:147138 HCAPLUS
DOCUMENT NUMBER: 92:147138
TITLE: Moranoline derivatives
INVENTOR(S): Matsumura, S.; Enomoto, H.; Aoyagi, Y.; Yoshikuni, Y.; Kura, K.; Yagi, M.; Shirahase, I.
PATENT ASSIGNEE(S): Nippon Shinyaku Co., Ltd., Japan
SOURCE: Belg., 39 pp.
CODEN: BEXXAL
DOCUMENT TYPE: Patent
LANGUAGE: French
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| BE 876020 | A1 | 19790903 | BE 1979-194978 | 19790503 |
| JP 54145672 | A2 | 19791114 | JP 1978-53603 | 19780503 |
| JP 59043947 | B4 | 19841025 | | |
| JP 55009051 | A2 | 19800122 | JP 1978-82606 | 19780706 |
| JP 59043948 | B4 | 19841025 | | |
| JP 55047655 | A2 | 19800404 | JP 1978-120661 | 19780929 |
| JP 59043949 | B4 | 19841025 | | |
| JP 55098163 | A2 | 19800725 | JP 1979-5714 | 19790120 |
| JP 60026387 | B4 | 19850624 | | |
| AT 8102785 | A | 19821115 | AT 1981-2785 | 19810623 |
| AT 371440 | B | 19830627 | | |

PRIORITY APPLN. INFO.:

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|---|---|
| JP 1978-53603 | 19780503 |
| JP 1978-82606 | 19780706 |
| JP 1978-120661 | 19780929 |
| JP 1979-5714 | 19790120 |
| AT 1979-3247 | 19790430 |

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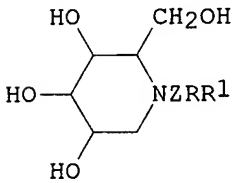


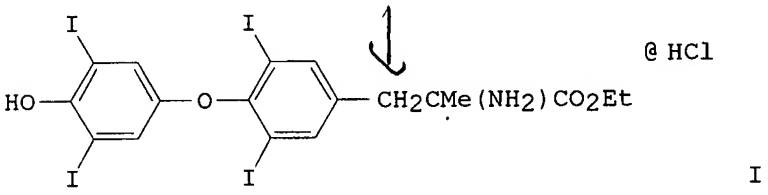

I

AB Moranoline derivs. I (Z = aliph. chain optionally contg. double and/or

triple bonds; R = Ph, substituted Ph, thienyl, 1,3-benzodioxol-5-yl; R1 = H, Ph, substituted phenyl) and their acid addn. salts, with antidiabetic activity (extensive data given), were prepd. from moranoline. Thus, moranoline was treated with Ph(CH2)4Br to give I [ZRR1 = Ph(CH2)4].

IT **73244-09-4P**
RL: BAC (Biological activity or effector, except adverse); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. and **antidiabetic** activity of)

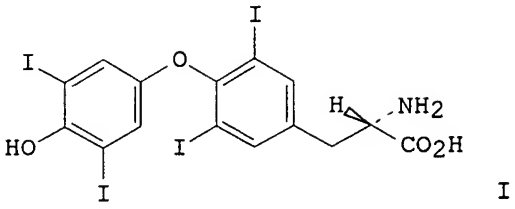
L12 ANSWER 51 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1979:180699 HCAPLUS
DOCUMENT NUMBER: 90:180699
TITLE: Experimental animal studies with a new lipid lowering compound: etiroxate hydrochloride
AUTHOR(S): Beckmann, R.
CORPORATE SOURCE: Biochem. Abt., Chem. Gruenenthal G.m.b.H., Stolberg, Ger.
SOURCE: Arzneim.-Forsch. (1979), 29(3), 499-508
CODEN: ARZNAD; ISSN: 0004-4172
DOCUMENT TYPE: Journal
LANGUAGE: German
GI




AB Orally administered etiroxate-HCl (I) **[55327-22-5]** decreased serum cholesterol [57-88-5] and serum **triglycerides** at doses of 2.8 and .gtoreq. 10 mg/kg, resp., in hypercholesterolemic rats. I was not anticholesterolemic in normolipemic animals. The effect of I on O consumption, heart rate, and heart wt. as well as its antigoitrogenic effect were much less than those of L- and D-thyroxine. The **therapeutic** index, as calcd. from the ratio of the dose having an effect on basal metab. to the dose affecting serum cholesterol, was 10-35 for I and 1 for either of the thyroxines.

L12 ANSWER 52 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1978:573680 HCAPLUS
DOCUMENT NUMBER: 89:173680
TITLE: The influence of fasting, diabetes, and several pharmacological agents on the pathways of thyroxine metabolism in rat liver
AUTHOR(S): Balsam, Alan; Ingbar, Sidney H.; Sexton, Franklin
CORPORATE SOURCE: Thorndike Lab., Harvard Med. Sch., Boston, Mass., USA
SOURCE: J. Clin. Invest. (1978), 62(2), 415-24
CODEN: JCINAO; ISSN: 0021-9738

DOCUMENT TYPE: Journal
LANGUAGE: English
GI



AB As judged from both paper and column chromatog., slices or homogenates of liver from rats fasted for 48 h displayed a lesser rate of generation of 125I-labeled 3,5,3'-triiodothyronine (T3) [**6893-02-3**] from 125I-labeled thyroxine (I) [**51-48-9**], added to incubation media than did prepns. from normal chow-fed animals. A similar defect in the conversion of I to T3 in the livers of fasted animals was obsd. when prepns. were incubated with substrate concns. of I so that T3 generation could be assessed by radioimmunoassay. Diminished generation of T3 from I was evident in the livers of animals with streptozotocin-induced **diabetes** mellitus, and this defect was overcome by the provision of insulin in vivo, but not in vitro. Decreased formation of T3 from I was also obsd. in prepns. of liver from animals given dexamethasone Na phosphate [2392-39-4], amiodarone [1951-25-3], and propylthiouracil [51-52-5]. In no case could these effects on the net formation of T3 from I be explained by effects of the exptl. conditions on the degrdn. of the T3 generated as judged from the rate of degrdn. of exogenous 125-I-T3 measured in parallel incubates. Reverse T3 formation was actively proceeding in the prepns. studied, was slightly enhanced by fasting, was unaffected by dexamethasone and amiodarone, and was markedly inhibited by propylthiouracil. In view of the similarities between the effect of these exptl. manipulations on the generation of T3 from I by rat liver in vitro to their effects on the prodn. rates and serum concns. of T3 in man, it is concluded that the rat liver system provides a suitable model for the study of factors that influence the conversion of I to T3 in man. In addn., the findings strongly indicate that this process, at least in the liver, is closely linked to the utilization of carbohydrate.

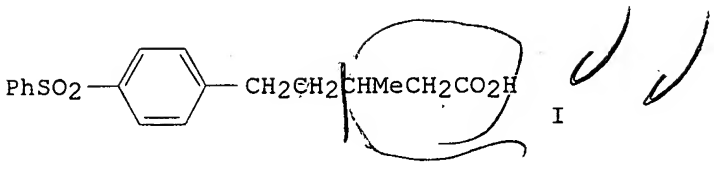
IT **6893-02-3**
RL: FORM (Formation, nonpreparative)
(formation of, from thryoxine, in liver, **diabetes** and fasting and **drugs** effect on)

IT **51-48-9**, biological studies
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(metab. of, by liver, **diabetes** and fasting and **drugs** effect on)

L12 ANSWER 53 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1978:485101 HCAPLUS
DOCUMENT NUMBER: 89:85101
TITLE: Effects of thyroid status on plasma adrenaline and

noradrenaline concentrations in sheep during acute and chronic cold exposure
AUTHOR(S): Christopherson, R. J.; Thompson, J. R.; Hammond, V. A.; Hills, G. A.
CORPORATE SOURCE: Dep. Anim. Sci., Univ. Alberta, Edmonton, Alberta, Can.
SOURCE: Can. J. Physiol. Pharmacol. (1978), 56(3), 490-6
CODEN: CJPPA3; ISSN: 0008-4212
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Plasma concns. of adrenaline (I) [51-43-4], noradrenaline (II) [51-41-2], and glucose were detd. in intact sheep and in surgically thyroidectomized sheep treated i.m. with either 0.125 or 0.25 mg triiodothyronine (T3) [**6893-02-3**]/day. On day 28 of exposure to temps. of 22-25 or 2-5.degree., overall mean plasma concns. of I were 0.07 and 0.15 ng/mL, resp., and of II were 0.30 and 0.45 ng/mL, resp. Plasma I concns. were higher in intact compared with thyroidectomized sheep on T3 **therapy**. Plasma glucose concns. were increased by exposure to 2-5.degree. and by T3 treatment. In a 2nd expt., thyroidectomized sheep were kept at 22-26.degree. and were either T3-treated (0.07 mg T3/day, i.m.) or untreated. After 3 wk, mean concns. in the untreated sheep before acute cold and during the last hour of cold exposure (-23.degree.) were, resp.: for I, 0.09 and 0.47 ng/mL; for II, 0.46 and 3.15 ng/mL; and for glucose, 62.1 and 122.1 mg/100 mL. In T3-treated sheep the mean concns. before and during cold were, resp.: for I, 0.07 and 0.22 ng/mL; for II, 0.30 and 1.71 ng/mL; and for glucose 59.6 and 82.3 mg/100 mL. The untreated sheep showed greater increases in plasma concns. of I, II, and glucose and in hematocrit values than T3-treated sheep, but had slightly smaller increases in metabolic rate, greater decreases in rectal temp., and similar heart rates during cold exposure.
IT **6893-02-3**
RL: BIOL (Biological study)
(catecholamines and **sugar** of **blood** plasma response to, cold in relation to)

L12 ANSWER 54 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1977:577451 HCAPLUS
DOCUMENT NUMBER: 87:177451
TITLE: Hypolipidemic activity of 5-aryl-3-methylvaleric acid derivatives
AUTHOR(S): Dygos, John H.; Jett, Charlene M.; Chinn, Leland J.; Miller, James E.
CORPORATE SOURCE: Dep. Chem. Res., Searle Lab., Chicago, Ill., USA
SOURCE: J. Med. Chem. (1977), 20(12), 1705-8
CODEN: JMCMAR
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



I

AB Seven title compds. were prepd. by Friedel-Crafts acylation of the appropriate arom. or heterocyclic deriv. with 4-chlorocarbonyl-3-methylbutanoic acid Me ester [56889-46-4] followed by hydrolysis of the ester and a modified Wolff-Kishner redn. Most of the compds. were active in lowering serum cholesterol levels in rats, and all compds. reduced serum **triglyceride** levels. 5-(4-Phenylsulfonylphenyl)-3-methylvaleric acid (I) [**64157-63-7**] was the most active compd., and lowered serum cholesterol levels 45% and serum **triglyceride** levels 60%. Structure-activity relations are discussed.

L12 ANSWER 55 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1977:527856 HCAPLUS
DOCUMENT NUMBER: 87:127856
TITLE: Effects of thyroid hormone (triiodothyronine and thyroxine) on blood pressure, heart rate and electrocardiographic changes in heart in cold
AUTHOR(S): Das Gupta, S.; Lahiri, P.; Roy, Bijon
CORPORATE SOURCE: Dep. Biophys., Sch. Trop. Med., Calcutta, India
SOURCE: Indian J. Cryog. (1976), 1(1), 71-4
CODEN: IJCRDD
DOCUMENT TYPE: Journal
LANGUAGE: English
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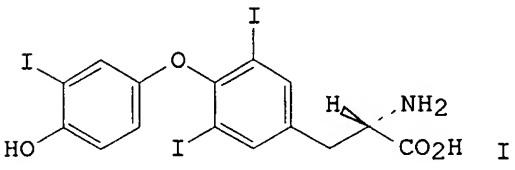


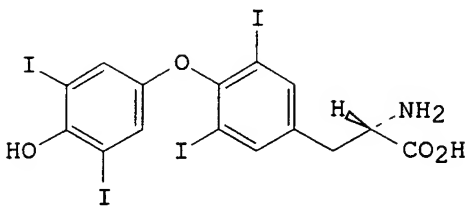
I

AB In anesthetized cats, cooling the body from the normal body temp. of 36.degree. to 24.degree. caused a progressive fall in arterial **blood pressure** and heart rate. In addn., alterations in the electrocardiogram obsd. included an increase in P-R, P-Q, and QRS intervals and lengthening of Q-T, S-T, and T-P segments with a max. effect on QT. There was also an elevation of the early part of S-T segment with onset of J wave and inversion of T wave. In some cases arrhythmias, bradycardia, complete heart block, and ventricular fibrillation occurred. O administration reversed cold-induced inverted T wave and delay in the onset of the J wave. Administration of triiodothyronine (I) [**6893-02-3**], but not of thyroxine [**51-48-9**], caused an increase in systemic arterial pressure, an increase in heart rate, reversion of flat or inverted T waves, and a decrease in the height of J

wave. The results are discussed in relation to I **therapy** of hypothermic coma.

L12 ANSWER 56 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1977:133278 HCAPLUS
DOCUMENT NUMBER: 86:133278
TITLE: Vascular toxicity of drugs: an accelerated and quantitative technique of assessment
AUTHOR(S): Sterne, J.; Brohon, J.
CORPORATE SOURCE: Res. Dep., SNELA, Suresnes, Fr.
SOURCE: Proc. Eur. Soc. Toxicol. (1976), 17(Predict. Chronic Toxic. Short Term Stud., Proc. Meet., 1975), 198-202
CODEN: PESTD5
DOCUMENT TYPE: Journal
LANGUAGE: English
AB To assess the effect of **drugs** on the arterial wall, rabbits were treated for 5 days with epinephrine [51-43-4] and thyroxine [**51-48-9**] and then maintained for 15 days on a high-fat diet. The rabbits developed aortic lesions showing all the degrees of atherosclerosis in man, from the single swelling of the endothelium to the atherosclerotic plaque with its fatty infiltration and evolution towards calcification. These lesions were quant. and statistically assessable. The lipid content in the whole aorta also was measured. Simultaneously, to compare sep. the effect on blood lipids, rats, which do not develop aortic lesions, are fed with a special high-fat diet and blood lipids are measured after 15 days. Clofibrate [637-07-0] decreased in blood lipids but had no effect on aorta damage or aorta lipids. Metformin [657-24-9] treatment did not alter the blood lipids of the rat but did decrease **triglycerides** in the rabbit aorta.
IT **51-48-9**, biological studies
RL: BIOL (Biological study)
(in artery response to **drugs** detn.)

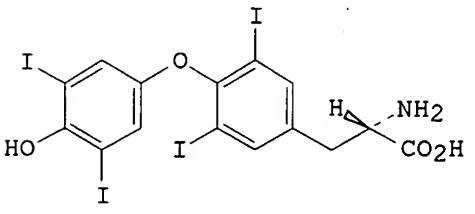
L12 ANSWER 57 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1977:115570 HCAPLUS
DOCUMENT NUMBER: 86:115570
TITLE: Urinary excretion of carnitine in patients with hyperthyroidism and hypothyroidism: augmentation by thyroid hormone
AUTHOR(S): Maebashi, M.; Kawamura, N.; Sato, M.; Imamura, A.; Yoshinaga, K.; Suzuki, M.
CORPORATE SOURCE: Sch. Med., Tohoku Univ., Sendai, Japan
SOURCE: Metab., Clin. Exp. (1977), 26(4), 351-6
CODEN: METAAJ
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



I

AB Urinary excretion of carnitine [541-15-1] and serum concns. of carnitine, **triglyceride**, and free fatty acids were measured in 54 hyperthyroid and 13 hypothyroid patients. In hyperthyroid patients urinary excretion of carnitine was increased above that of the control subjects. On adequate treatment with antithyroid **drug**, carnitine excretion was reduced to the normal range, and serum lipids changed in parallel. In contrast, carnitine excretion was markedly reduced in hypothyroid patients. After substitution **therapy** with thyroid hormones the excretion increased in these patients. This change was assocd. with a marked decrease of serum **triglyceride**. There was an inverse correlation between urinary excretion of carnitine and serum **triglyceride** concn. Carnitine excretion was correlated with serum thyroxine (I) [**51-48-9**] concn. in hyper- and hypothyroid patients. Apparently, thyroid hormones play an important role in carnitine metab., which in turn influences serum **triglyceride** metab.

L12 ANSWER 58 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1976:572176 HCAPLUS
DOCUMENT NUMBER: 85:172176
TITLE: Reversal of decreased human adipose tissue lipoprotein lipase and hypertriglyceridemia after treatment of hypothroidism
AUTHOR(S): Pykalisto, Olavi; Goldberg, Andrew P.; Brunzell, John D.
CORPORATE SOURCE: Sch. Med., Univ. Washington, Seattle, Wash., USA
SOURCE: J. Clin. Endocrinol. Metab. (1976), 43(3), 591-600
CODEN: JCEMAZ
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



I

AB Lipoprotein lipase (LPL) [9004-02-8] was measured in the subcutaneous adipose tissue of 6 hypothyroid patients before and during **therapy** with L-thyroxine (I) [**51-48-9**]. The activity of the activated form of the enzyme, measured as heparin elutable LPL, was lower in hypothyroid patients (1.54 munits/106 cells) than in controls (3.26) and increased (163%) with treatment to levels comparable to the controls. The total activity of LPL was in the low normal range in the hypothyroid patients (0.68), but not significantly different from normal (1.10) and did not increase significantly with treatment. Plasma post heparin lipolytic activity (PHLA) was low in hypothyroidism and increased (111%) with treatment. These increases in PHLA correlated with the increases in the activity of heparin elutable LPL. In all patients, fasting plasma **triglyceride** levels decreased (-43%) after treatment. Serial detn. of heparin elutable LPL activity, PHLA, and plasma **triglyceride** during I treatment revealed a correlation between the per cent changes in PHLA and heparin elutable LPL activity, an inverse correlation between plasma **triglyceride** levels and heparin elutable LPL, and no correlation between plasma **triglyceride** and PHLA. Apparently, the low PHLA and **hypertriglyceridemia** of hypothyroidism are related to low adipose tissue LPL activity.

L12 ANSWER 59 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1976:472229 HCAPLUS
DOCUMENT NUMBER: 85:72229
TITLE: Effect of nitrogen-containing derivatives of 1,4-dicarboxylic acids on plasma cholesterol concentration in rats
AUTHOR(S): Yen, M. S.; Chow, S. Y.
CORPORATE SOURCE: Inst. Biophys., Natl. Def. Med. Cent., Taipei, Taiwan
SOURCE: Chung-Hua I Hsueh Tsa Chi (Taipei) (1975), 22(4), 237-41
CODEN: CIHCDM
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Of 9 nitrogen-contg. derivs. of 1,4-dicarboxylic acids to maleamic acid derivs., N-(p-methylphenyl)maleamic acid [24870-11-9] and N-adamantylmaleamic acid [54395-92-5] lowered the plasma cholesterol level in normal and propylthiouracil-induced hypercholesteremic rats. These compds. also slightly reduced the plasma contents of **triglycerides** and phospholipids in normal rats. The effects are comparable to those of triiodothyronine [**6893-02-3**].

L12 ANSWER 60 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1976:159631 HCAPLUS
DOCUMENT NUMBER: 84:159631
TITLE: Effects of various hypolipidemic drugs on fatty acid composition of liver and serum lipids
AUTHOR(S): Maier, Rene; Muller, Klaus
CORPORATE SOURCE: Pharm. Div., Ciba-Geigy Ltd., Basel, Switz.
SOURCE: Adv. Exp. Med. Biol. (1975), 63(Lipids, Lipoproteins, Drugs), 349-57
CODEN: AEMBAP
DOCUMENT TYPE: Journal
LANGUAGE: English

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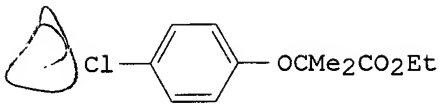

I

AB In rats, treatment with the aryloxy fatty acids, clofibrate (I) [637-07-0] and C 13437-Su (Nafenopine) [3771-19-5] increased the oleic acid [112-80-1] content and decreased the linoleic acid [60-33-3] content of serum and liver cholesterol esters, **triglycerides**, and phospholipids. The effect was dose-dependent starting at doses eliciting hypolipidemic effects. L-thyroxine [**51-48-9**] or nicotinic acid [59-67-6] did not affect the fatty acid levels. Possible mechanisms of action of the hypolipidemic **drugs** are discussed.

L12 ANSWER 61 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1975:38909 HCAPLUS
DOCUMENT NUMBER: 82:38909
TITLE: Thyroidectomy and combined thyroxine-cortisol therapy. Their effects on blood sugar, serum immunoreactive insulin, and free fatty acids during an oral glucose tolerance test
AUTHOR(S): Renauld, Aurora; Sverdlik, Rita C.
CORPORATE SOURCE: Fac. Med., Univ. Buenos Aires, Buenos Aires, Argent.
SOURCE: Acta Diabetol. Lat. (1974), 11(2), 96-105
CODEN: ADILAS
DOCUMENT TYPE: Journal
LANGUAGE: English

AB **Blood sugar**, serum immunoreactive insulin [9004-10-8] and free fatty acid responses to glucose [50-99-7] stimuli during an oral glucose tolerance test in dogs in 3 exptl. conditions (normality, untreated and thyroxine [**51-48-9**]-cortisol [50-23-7] treated hypothyroidism) have been studied. The basal levels of **blood sugar**, serum immunoreactive insulin and free fatty acids remained unaffected by either the untreated or treated hypothyroid conditions. Thyroidectomy delayed both the onset of hyperglycemia following the oral **glucose** load and the return of **blood sugar** levels to baseline at the end of the test; these changes were completely reversible by the combined **therapy**. The grossly exaggerated serum insulin response to hyperglycemia in dogs after thyroidectomy was markedly decreased but not normalized by the combined **therapy**. Glucose induced a prompt and marked drop of the serum free fatty acid levels in the normal dogs, followed by a recovery period at the end of the test. In hypothyroid dogs, the fall was slower and less intense, and the recovery period was not obsd. Combined thyroxine-cortisol administration improved only the recovery period, and the improvement was only partial. The foregoing results show the importance of the effects of both hypothyroidism itself and of secondary adrenocortical deficiency for the development of the disturbances of intestinal glucose absorption and insulin response to hyperglycemia found in dogs after thyroidectomy.

L12 ANSWER 62 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1974:91530 HCAPLUS
DOCUMENT NUMBER: 80:91530
TITLE: Effect of oral contraceptives on biochemistry normal values
AUTHOR(S): Wilson, Leiana M.
CORPORATE SOURCE: St. Paul's Hosp., Vancouver, B. C., Can.
SOURCE: Can. J. Med. Technol. (1973), 35(6), 42,44-6,51-4,57-8
CODEN: CJMTAY
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In women taking oral contraceptives (Ortho-Novum [8015-29-0], Ovral [8056-51-7]) mean values of serum .alpha.1-globulin, .alpha.2-globulin, thyroxine [**51-48-9**], and cortisol [50-23-7] were higher and albumin, calcium [7440-70-2], and triiodothyronine [**6893-02-3**] were lower than in women not taking the **drugs**. Serum glutamic-oxalacetic transaminase, lactate dehydrogenase, lipids, total protein, .beta.-globulin, .gamma.-globulin, bilirubin, alk. phosphatase, fasting **blood sugar**, **blood** urea N, CO2, Cl-, K, Na, uric acid, P, and creatinine were not significantly affected by the contraceptives.

L12 ANSWER 63 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1974:22995 HCAPLUS
DOCUMENT NUMBER: 80:22995
TITLE: Effect of thyroid hormone on cerebral glucose metabolism in the infant rat
AUTHOR(S): Moore, Thomas J.; Lione, Armand P.; Regen, David M.
CORPORATE SOURCE: Coll. Physicians Surg., Columbia Univ., New York, N. Y., USA
SOURCE: Amer. J. Physiol. (1973), 225(4), 925-9
CODEN: AJPHAP
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Transport rate of **sugar** across the **blood**-brain barrier in newborn rats was one-fourth that of the adult rate until 12 days of age at which time it began to increase over the next 8 days to adult levels. Daily injections of L-thyroxine (I) [**51-48-9**] (1 mg/kg) caused the transport rate to rise earlier, whereas the antithyroid **drug**, methimazole [60-56-0], delayed the rise in transport activity.

L12 ANSWER 64 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1973:474074 HCAPLUS
DOCUMENT NUMBER: 79:74074
TITLE: Ether derivatives of a progestin [progesterone] and estrogen in monthly dosage
AUTHOR(S): Danowski, Thaddeus S.; Wilson, H. Randolph; Vester, John W.; Fisher, Edwin R.; Khurana, Ramesh C.; Noland, Sean; Stephan, Thorsten; Sunder, Joseph H.
CORPORATE SOURCE: Dep. Med., Univ. Pittsburgh, Pittsburgh, Pa., USA
SOURCE: Clin. Pharmacol. Ther. (1973), 14(3), 455-61
CODEN: CLPTAT
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Menopausal women receiving oral doses of quingestanol acetate-quinestrol mixt. [39356-32-6] (2.5 mg and 2 mg, resp.) at 3-4 week intervals for a yr. showed increased nos. of superficial cells in vaginal smears and decreases in intermediate and parabasal cells. The **medication** normalized serum FSH [9002-68-0] and LH [9002-67-9] which had increased during menopause. Serum **triglyceride** and thyroxine [**51-48-9**] and plasma 11-hydroxycorticosteroids were consistantly increased. Serum inorg. phosphorus [7723-14-0] and calcium [7440-70-2] decreased. Urinary steroids and their response to oral metyrapone were unchanged.

L12 ANSWER 65 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1973:106327 HCAPLUS
DOCUMENT NUMBER: 78:106327
TITLE: Effect of cortisol-thyroxine combined therapy on the insulin response to hyperglycemia in thyroidectomized dogs
AUTHOR(S): Renauld, Aurora; Sverdlik, Rita C.
CORPORATE SOURCE: Fac. Med., Univ. Buenos Aires, Buenos Aires, Argent.
SOURCE: Acta Physiol. Lat. Amer. (1972), 22(4), 251-7
CODEN: APLTAF
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In thyroidectomized dogs, the elevated **blood sugar** and serum insulin [9004-10-8] in hyperglycemia, induced by rapid glucose infusion (1 g/kg, for 1 min), were normalized by treatment with cortisol [50-23-7] (1 mg/kg/day, for 13 days, s.c.) and thyroxine (I) [**51-48-9**] (10 .mu.g/kg/day, for 10 days). The depressed serum fatty acid levels after glucose loading in thyroidectomized dogs failed to respond to cortisol and I **therapy**. Evidently, the overnormal stimulatory effects of cortisol on insulin secretion in hyperglycemic thyroidectomized dogs is due to the absence of the inhibitory influence of the thyroid gland.

L12 ANSWER 66 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1972:429006 HCAPLUS
DOCUMENT NUMBER: 77:29006
TITLE: Comparison of hypolipidemic drugs in the prevention of an orotic acid fatty liver
AUTHOR(S): Elwood, J. Clint; Richert, Dan A.; Westerfeld, W. W.
CORPORATE SOURCE: Upstate Med. Cent., State Univ. New York, Syracuse, N. Y., USA
SOURCE: Biochem. Pharmacol. (1972), 21(8), 1127-34
CODEN: BCPCA6
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Et p-chlorophenoxyisobutyric acid (I) [637-07-0], U 22105 [p-phenoxyphenyl methanesulfonate] [23419-81-0], U 25030 [N,N-dimethyl-N'-(4-phenoxyphenyl)sulfamide] [23419-78-5], and SQ 11071 [2,2'''-[(1-methyl-4,4-diphenylbutylidene)bis(p-phenyleneoxy)]bis(triethylamine) citrate] [26718-22-9] had approx. the same activity in preventing fatty liver in rats when added to a 1% orotic acid [65-86-1] diet. 1-Methyl-4-piperidyl bis(p-chlorophenoxy)acetate (II) [22204-91-7] was 2-3 times as active, and Su 13437 [2-methyl-2-(p-1,2,3,4-tetrahydro-1-naphthylphenoxy)propionic

acid](III) [3771-19-5] was 10 times as active as I. On the same wt. basis, Choloxin [**137-53-1**] was 80-90 times as active. Dilantin [630-93-3], L-thyroxine [**51-48-9**], allopurinol [315-30-0], and 5,5-diphenyl-2-thiohydantoin [21083-47-6] were as active as most of the **drugs** in preventing **triglyceride** deposition, but appreciably higher concns. of these compds. were needed to prevent cholesterol [57-88-5] deposition. I, II, III, Choloxin, and L-thyroxine increased liver .alpha.-glycerophosphate dehydrogenase [9001-49-4] activity. Lipotropic effect of these **drugs** was not mediated through this enzyme. Feeding orotic acid alone practically eliminated the pre-.beta. and .beta.-lipoprotein (.beta.LP) band from the serum gel electrophoresis, but the intensity of the .beta. band was restored by the active **drugs** in proportion to their dosages; inactive substances did not restore the .beta.LP band. A quick screening procedure was developed for substances which intensify the serum .beta.LP band.

L12 ANSWER 67 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1972:414655 HCAPLUS
DOCUMENT NUMBER: 77:14655
TITLE: Studies on the effect of thyroxine on in vivo insulin secretion as modified by hypophysectomy
AUTHOR(S): Renauld, Aurora; Pinto, Jorge E. B.; Sverdlik, Rita C.; Foglio, Virgilio G.; Pallotta, M. G.; Carrera Vescio, Luis
CORPORATE SOURCE: Fac. Med., Univ. Buenos Aires, Buenos Aires, Argent.
SOURCE: Diabetologia (1972), 7(6), 445-8
CODEN: DBTGAJ
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Thyroxine (I) [**51-48-9**] (0.5 or 100 .gamma./kg/day, for 10 days) elevated back to normal **blood sugar** levels in hypophysectomized dogs, but had no effect on serum immunoreactive insulin [9004-10-8]. The rate of disappearance of **glucose** [50-99-7] from the **blood** was normal in the hypophysectomized dogs and was unaffected by I **therapy**. The increase in **blood sugar** during i.v. **glucose** tolerance test after hypophysectomy was corrected by I at 0.5 .gamma.; I at 100 .gamma., however, induced a further increase.

L12 ANSWER 68 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1972:400575 HCAPLUS
DOCUMENT NUMBER: 77:575
TITLE: Triton-induced hyperlipidemia in rats as an animal model for screening hypolipidemic drugs
AUTHOR(S): Schurr, P. E.; Schultz, J. R.; Parkinson, T. M.
CORPORATE SOURCE: Res. Lab., Upjohn Co., Kalamazoo, Mich., USA
SOURCE: Lipids (1972), 7(1), 68-74
CODEN: LPDSAP
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A screening test was described for hypolipidemic agents in which compds. are administered orally to fasted rats after a single i.v. injection of 225 mg Triton WR-1339 [25301-02-4]/kg, and serum cholesterol [57-88-5] and **triglycerides** are measured 43 hr post-Triton. Conditions for the

screen were established by studying interrelations between serum cholesterol, **triglycerides**, and Triton levels during the post-Triton period and the effects of the Triton dose, route of administration and fasting on serum lipid levels, and **drug** hypocholesterolemic activity. The test detects compds. which inhibit lipid biosynthesis or stimulate lipid catabolism. Several **drugs** with different mechanisms of action which are hypolipidemic in man, including nicotinic acid [59-67-6], D-thyroxine [**51-49-0**], triparanol [78-41-1], nafoxidine-HCl [1847-63-8], and clofibrate [637-07-0], are active in this system. Results with std. hypolipidemic **drugs** are reproducible and comform well to performance levels of the screen predicted from statistical anal.

L12 ANSWER 69 OF 69 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1968:46773 HCAPLUS
DOCUMENT NUMBER: 68:46773
TITLE: Effect of thyroid hormones on glucose tolerance in normals and diabetics
AUTHOR(S): El-Ridi, Mohamed S.; Higazi, Abdel M.; Ismail, Ahmed A.; Lotfi, R. H.; Fayek, K. I.; Talaat, Mohamed
CORPORATE SOURCE: Cairo Fac. Med., Cairo Univ., Cairo, Egypt
SOURCE: J. Egypt. Med. Assoc. (1967), 50(4-5), 233-44
CODEN: JEMAAJ
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Expts. with 5 normal and 30 mildly diabetic persons showed that the administration of L-thyroxine and L-triiodothyronine in moderate doses did not materially influence glucose tolerance, indicating that these compds. can be used in diabetics if necessary. 26 references.
IT **51-48-9**, biological studies **6893-02-3**
RL: BIOL (Biological study)
(in **diabetes therapy**, glucose tolerance in relation to)

=> select hit rn l12 1-69
E1 THROUGH E58 ASSIGNED

=> fil reg
FILE 'REGISTRY' ENTERED AT 11:17:53 ON 08 AUG 2002
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.


STRUCTURE FILE UPDATES: 7 AUG 2002 HIGHEST RN 443093-92-3
DICTIONARY FILE UPDATES: 7 AUG 2002 HIGHEST RN 443093-92-3

TSCA INFORMATION NOW CURRENT THROUGH January 7, 2002

Please note that search-term pricing does apply when conducting SmartSELECT searches.

Crossover limits have been increased. See HELP CROSSOVER for details.

Calculated physical property data is now available. See HELP PROPERTIES for more information. See STNote 27, Searching Properties in the CAS Registry File, for complete details:
http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf

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=> d ide can l3 1-58

L3 ANSWER 1 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 442906-46-9 REGISTRY
CN INDEX NAME NOT YET ASSIGNED
MF C26 H31 S . C2 F5 O3 S
SR CA
LC STN Files: CAPLUS

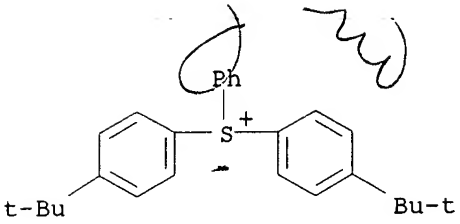
CM 1

CRN 108410-37-3
CMF C2 F5 O3 S

$^{-}O_3S-CF_2-CF_3$

CM 2

CRN 97878-76-7
CMF C26 H31 S

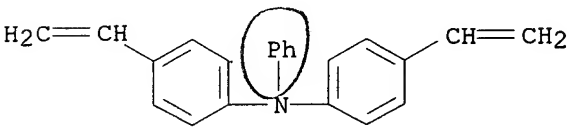



1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L3 ANSWER 2 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 442904-29-2 REGISTRY
CN INDEX NAME NOT YET ASSIGNED
MF (C22 H19 N)x
CI PMS
PCT Polystyrene
SR CA
LC STN Files: CAPLUS

CM 1

CRN 442904-28-1
CMF C22 H19 N




1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L3 ANSWER 3 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 442904-28-1 REGISTRY
CN INDEX NAME NOT YET ASSIGNED
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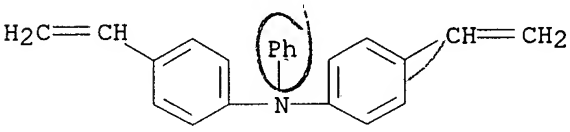
CI COM
SR CA
LC STN Files: CAPLUS

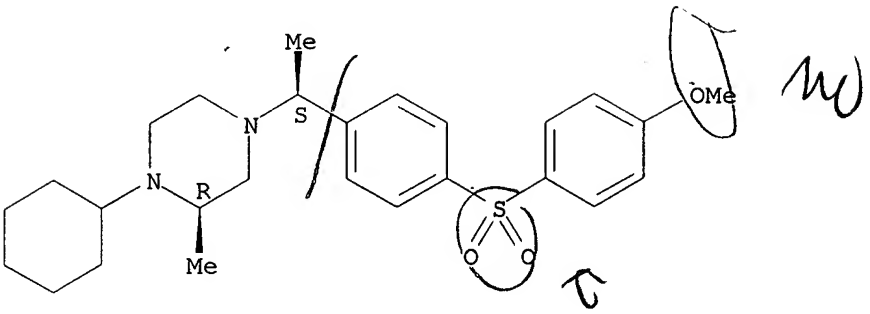



1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L3 ANSWER 4 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 442868-93-1 REGISTRY

CN INDEX NAME NOT YET ASSIGNED
FS STEREOSEARCH
MF C26 H36 N2 O3 S
SR CA
LC STN Files: CAPLUS

Absolute stereochemistry.

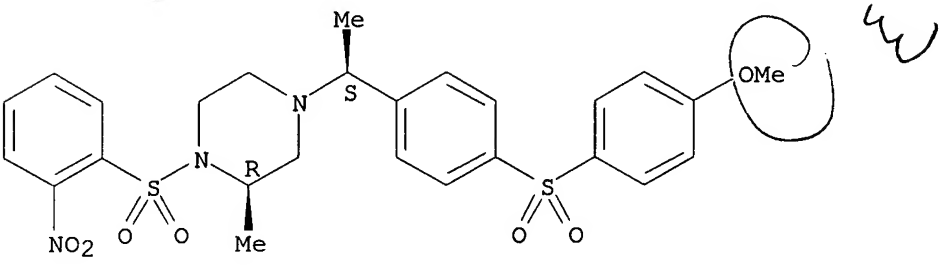



**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L3 ANSWER 5 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 442868-92-0 REGISTRY
CN INDEX NAME NOT YET ASSIGNED
FS STEREOSEARCH
MF C26 H29 N3 O7 S2
SR CA
LC STN Files: CAPLUS

Absolute stereochemistry.



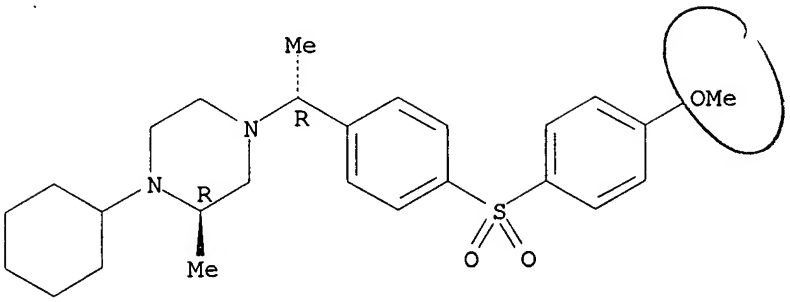

**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L3 ANSWER 6 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 442868-81-7 REGISTRY
CN INDEX NAME NOT YET ASSIGNED

FS STEREOSEARCH
MF C26 H36 N2 O3 S
SR CA
LC STN Files: CAPLUS

Absolute stereochemistry.

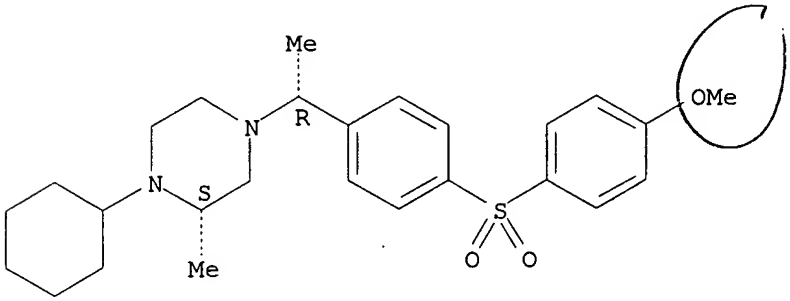



**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L3 ANSWER 7 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 442868-80-6 REGISTRY
CN INDEX NAME NOT YET ASSIGNED
FS STEREOSEARCH
MF C26 H36 N2 O3 S
SR CA
LC STN Files: CAPLUS

Absolute stereochemistry.

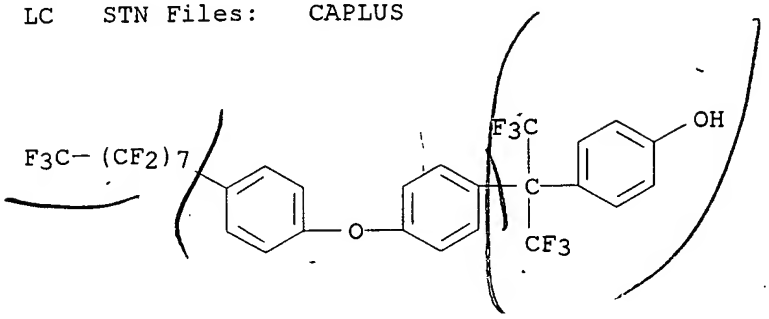



**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L3 ANSWER 8 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 442682-66-8 REGISTRY
CN Phenol, 4-[2,2,2-trifluoro-1-[4-[4-(heptadecafluorooctyl)phenoxy]phenyl]-1-

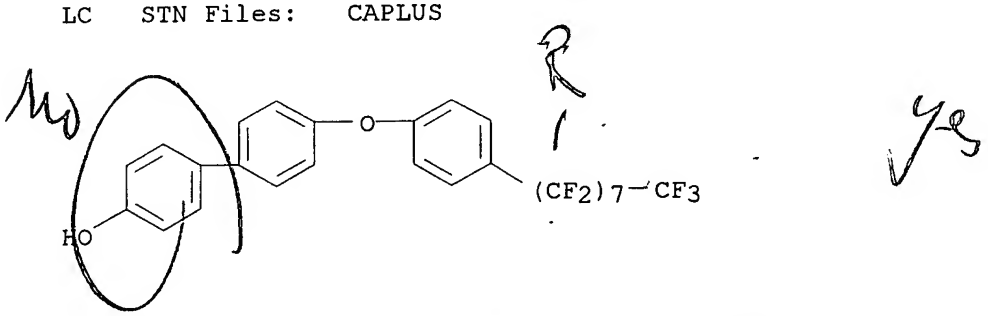
(trifluoromethyl)ethyl]- (9CI) (CA INDEX NAME)
MF C29 H13 F23 O2
SR CA
LC STN Files: CAPLUS




**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

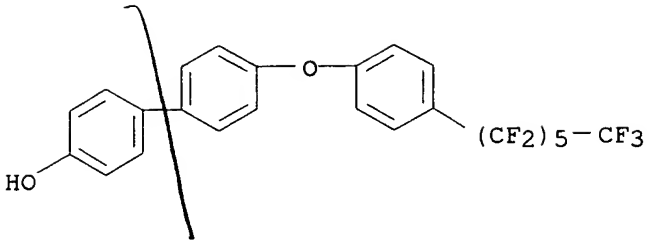
L3 ANSWER 9 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 442682-64-6 REGISTRY
CN [1,1'-Biphenyl]-4-ol, 4'-[4-(heptadecafluorooctyl)phenoxy]- (9CI) (CA INDEX NAME)
MF C26 H13 F17 O2
SR CA
LC STN Files: CAPLUS




**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

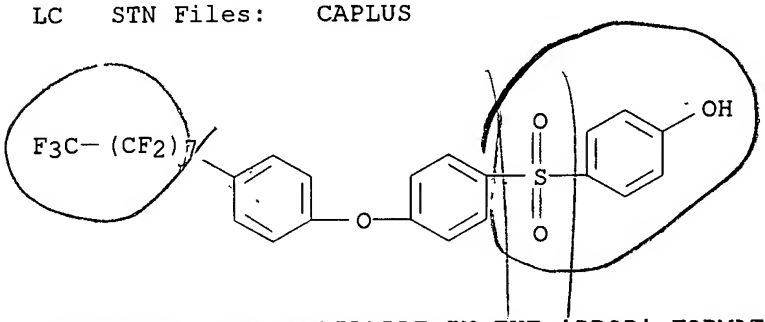
L3 ANSWER 10 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 442682-63-5 REGISTRY
CN [1,1'-Biphenyl]-4-ol, 4'-[4-(tridecafluorohexyl)phenoxy]- (9CI) (CA INDEX NAME)
MF C24 H13 F13 O2
SR CA
LC STN Files: CAPLUS




**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

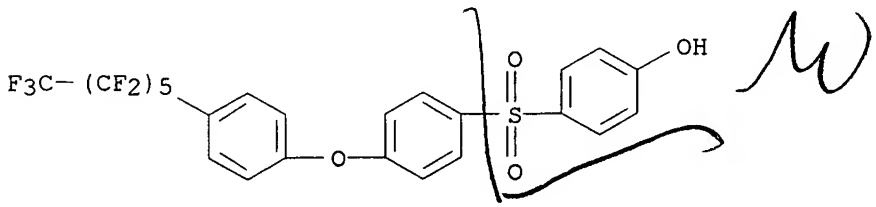
L3 ANSWER 11 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 442682-62-4 REGISTRY
CN Phenol, 4-[[4-[4-(heptadecafluorooctyl)phenoxy]phenyl]sulfonyl]- (9CI) (CA INDEX NAME)
MF C26 H13 F17 O4 S
SR CA
LC STN Files: CAPLUS




**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L3 ANSWER 12 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 442682-61-3 REGISTRY
CN Phenol, 4-[[4-[4-(tridecafluorohexyl)phenoxy]phenyl]sulfonyl]- (9CI) (CA INDEX NAME)
MF C24 H13 F13 O4 S
SR CA
LC STN Files: CAPLUS




**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L3 ANSWER 13 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 442655-41-6 REGISTRY
CN INDEX NAME NOT YET ASSIGNED
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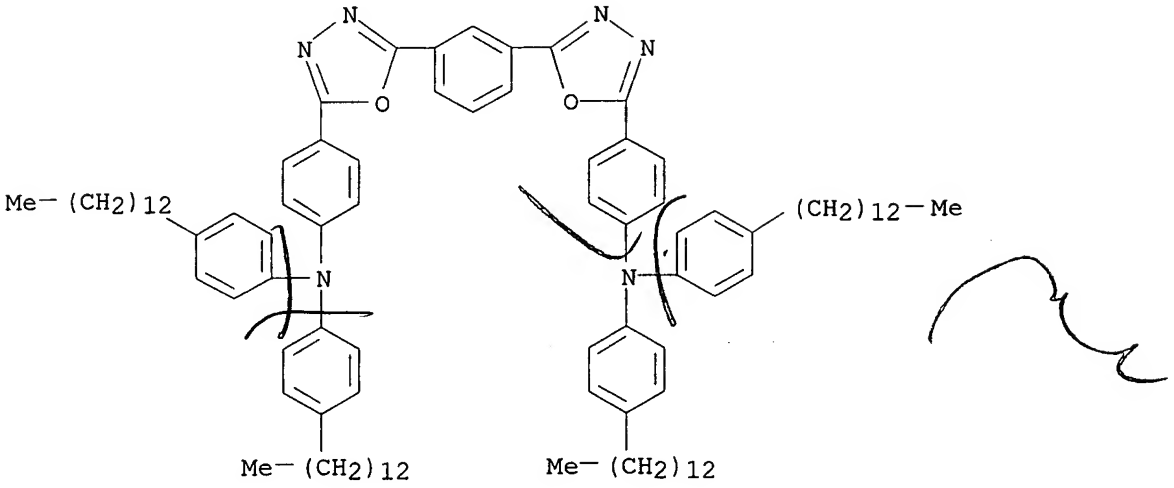
SR CA
LC STN Files: CAPLUS




1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L3 ANSWER 14 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 442655-40-5 REGISTRY
CN INDEX NAME NOT YET ASSIGNED
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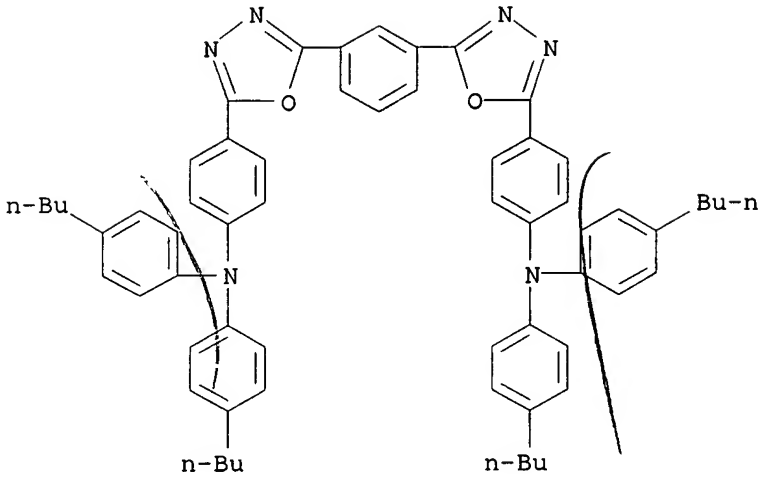
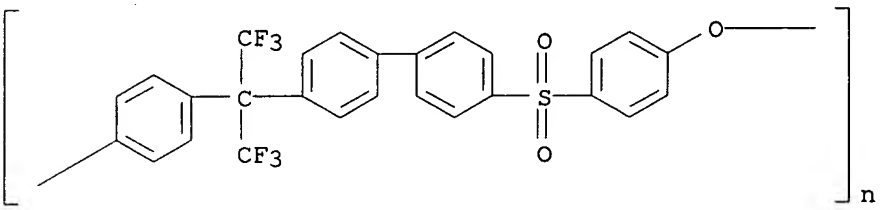
SR CA
LC STN Files: CAPLUS




**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L3 ANSWER 15 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 441776-16-5 REGISTRY
CN Poly[oxy-1,4-phenylenesulfonyl[1,1'-biphenyl]-4,4'-diyl[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene]-1,4-phenylene] (9CI) (CA INDEX NAME)
MF (C27 H16 F6 O3 S)n
CI PMS
PCT Polyether, Polysulfone
SR CA
LC STN Files: CAPLUS




1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L3 ANSWER 16 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 441312-74-9 REGISTRY
CN Carbonic acid, polymer with N,N-bis(4-methylphenyl)-4-[3-(triethoxysilyl)propyl]benzenamine, 4,4'-cyclohexylidenebis[phenol], 4,4'-cyclohexylidenebis[2-[3-(trimethoxysilyl)propyl]phenol] and trimethoxymethylsilane (9CI) (CA INDEX NAME)
MF (C30 H48 O8 Si2 . C29 H39 N O3 Si . C18 H20 O2 . C4 H12 O3 Si . C H2 O3)x

CI PMS
PCT Polycarbonate, Polycarbonate formed, Polyother
SR CA
LC STN Files: CAPLUS

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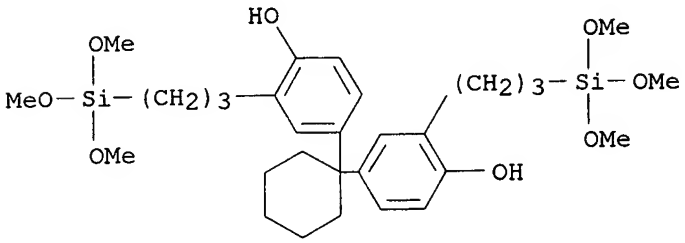
CRN 288373-15-9
CMF C30 H48 O8 Si2



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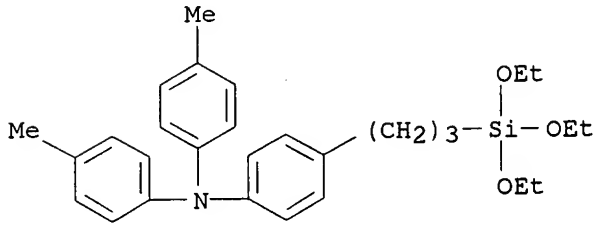
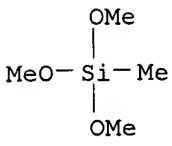
CRN 265658-50-2
CMF C29 H39 N O3 Si



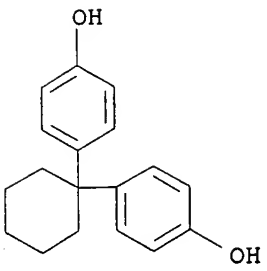
CM 3

CRN 1185-55-3
CMF C4 H12 O3 Si



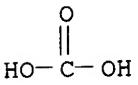
CM 4

CRN 843-55-0
CMF C18 H20 O2




CM 5

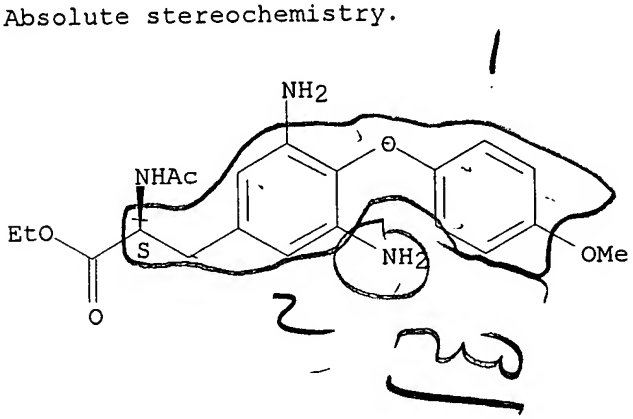
CRN 463-79-6
CMF C H2 O3




1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L3 ANSWER 17 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 440667-78-7 REGISTRY
CN L-Tyrosine, N-acetyl-3,5-diamino-O-(4-methoxyphenyl)-, ethyl ester (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C20 H25 N3 O5
SR CA

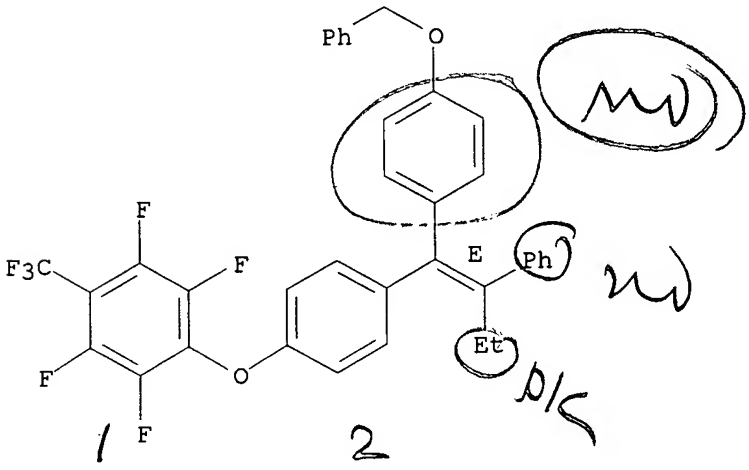
Absolute stereochemistry.



**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

L3 ANSWER 18 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 440646-13-9 REGISTRY
CN Benzene, 1-(phenylmethoxy)-4-[(1E)-2-phenyl-1-[4-[2,3,5,6-tetrafluoro-4-(trifluoromethyl)phenoxy]phenyl]-1-butenyl]- (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C36 H25 F7 O2
SR CA
LC STN Files: CA, CAPLUS

Double bond geometry as shown.



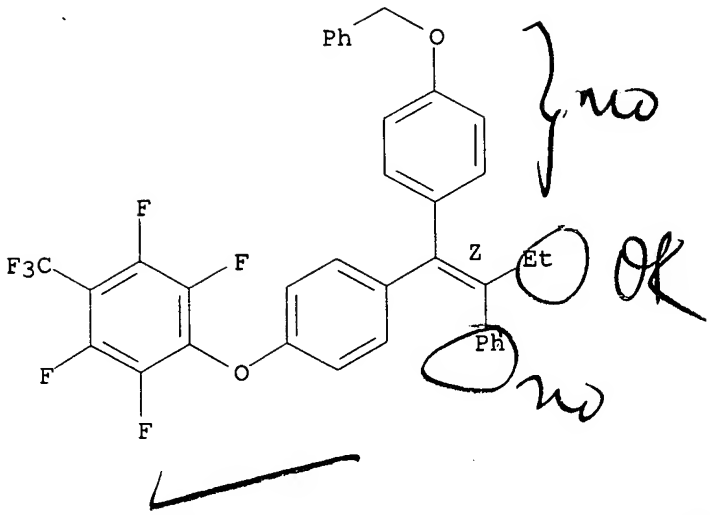

**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:78730

L3 ANSWER 19 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 440646-10-6 REGISTRY
CN Benzene, 1-(phenylmethoxy)-4-[(1Z)-2-phenyl-1-[4-[2,3,5,6-tetrafluoro-4-(trifluoromethyl)phenoxy]phenyl]-1-butenyl]- (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C36 H25 F7 O2
SR CA
LC STN Files: CA, CAPLUS

Double bond geometry as shown.

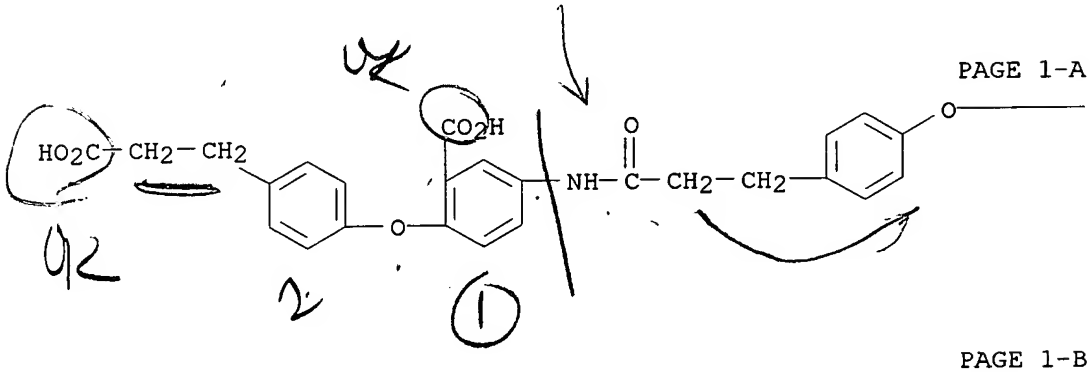



**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:78730

L3 ANSWER 20 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 440365-66-2 REGISTRY
CN Benzenepropanoic acid, 4-[2-carboxy-4-[[3-[4-(octadecyloxy)phenyl]-1-oxopropyl]amino]phenoxy]- (9CI) (CA INDEX NAME)
FS 3D CONCORD
MF C43 H59 N O7
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER



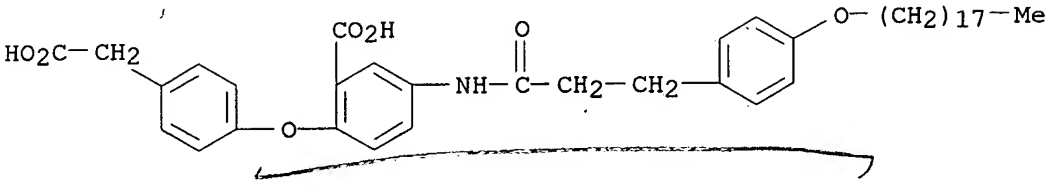

— $(CH_2)_{17}$ — Me

**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:73259

L3 ANSWER 21 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 440365-64-0 REGISTRY
CN Benzeneacetic acid, 4-[2-carboxy-4-[[3-[4-(octadecyloxy)phenyl]-1-oxopropyl]amino]phenoxy]- (9CI) (CA INDEX NAME)
FS 3D CONCORD
MF C42 H57 N O7
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER

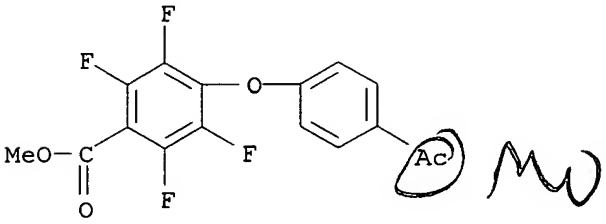


**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:73259

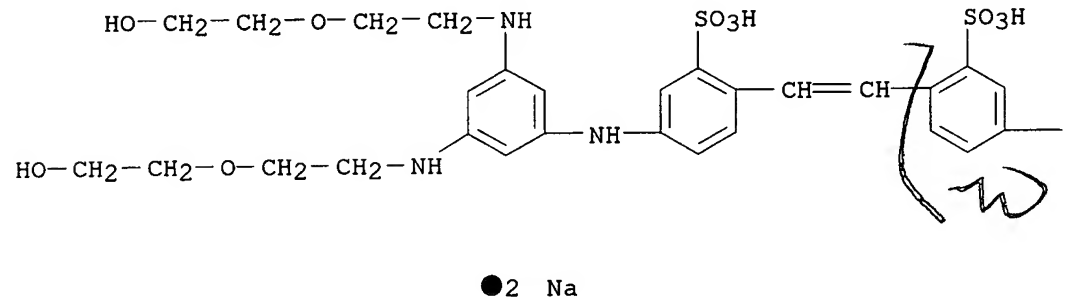
L3 ANSWER 22 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 440108-35-0 REGISTRY
CN Benzoic acid, 4-(4-acetylphenoxy)-2,3,5,6-tetrafluoro-, methyl ester (9CI) (CA INDEX NAME)
FS 3D CONCORD
MF C16 H10 F4 O4
SR Chemical Library



**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

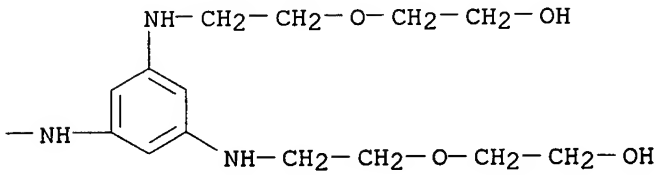
L3 ANSWER 23 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 439869-29-1 REGISTRY
CN Benzenesulfonic acid, 2,2'-(1,2-ethenediyl)bis[5-[[3,5-bis[[2-(2-hydroxyethoxy)ethyl]amino]phenyl]amino]-, disodium salt (9CI) (CA INDEX NAME)
MF C42 H58 N6 O14 S2 . 2 Na
SR CA
LC STN Files: CA, CAPLUS

PAGE 1-A



●2 Na

PAGE 1-B

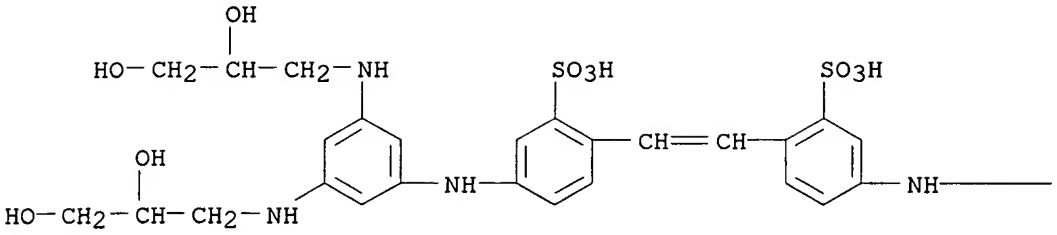


1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:70483

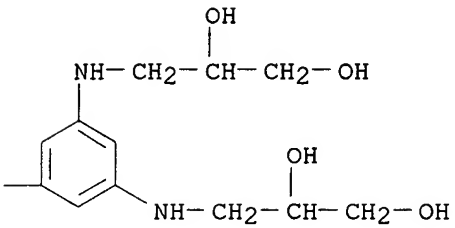
L3 ANSWER 24 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 439869-28-0 REGISTRY
CN Benzenesulfonic acid, 2,2'-(1,2-ethenediyl)bis[5-[[3,5-bis[(2,3-dihydroxypropyl)amino]phenyl]amino]-, disodium salt (9CI) (CA INDEX NAME)
MF C38 H50 N6 O14 S2 . 2 Na
SR CA
LC STN Files: CA, CAPLUS

PAGE 1-A



●2 Na

PAGE 1-B

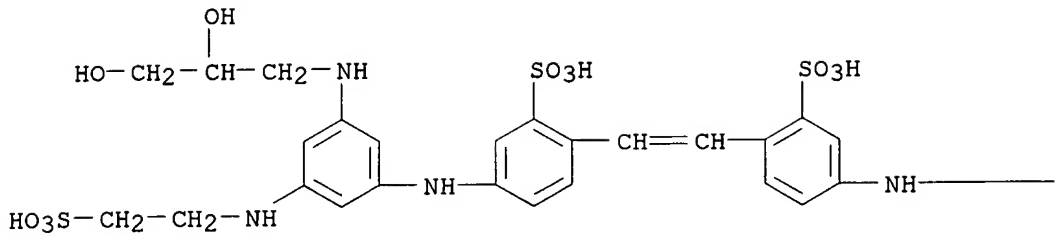


1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:70483

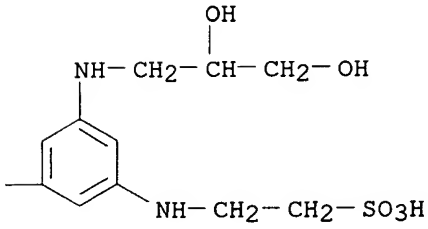
L3 ANSWER 25 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 439869-27-9 REGISTRY
CN Benzenesulfonic acid, 2,2'-(1,2-ethenediyl)bis[5-[[3-[(2,3-dihydroxypropyl)amino]-5-[(2-sulfoethyl)amino]phenyl]amino]-, hexasodium salt (9CI) (CA INDEX NAME)
MF C36 H46 N6 O16 S4 . 6 Na
SR CA
LC STN Files: CA, CAPLUS

PAGE 1-A



●6 Na

PAGE 1-B

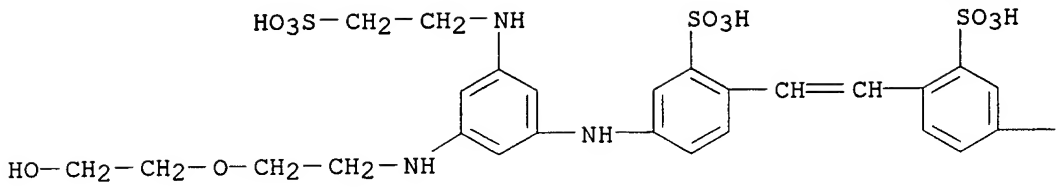


1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:70483

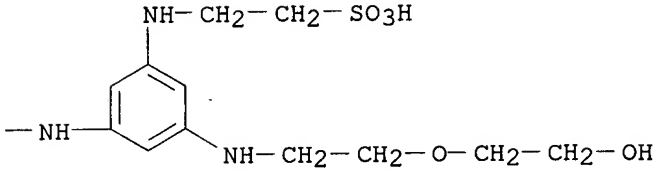
L3 ANSWER 26 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 439869-26-8 REGISTRY
CN Benzenesulfonic acid, 2,2'-(1,2-ethenediyl)bis[5-[[3-[[2-(2-hydroxyethoxy)ethyl]amino]-5-[(2-sulfoethyl)amino]phenyl]amino]-, tetrasodium salt (9CI) (CA INDEX NAME)
MF C38 H50 N6 O16 S4 . 4 Na
SR CA
LC STN Files: CA, CAPLUS

PAGE 1-A



●4 Na

PAGE 1-B

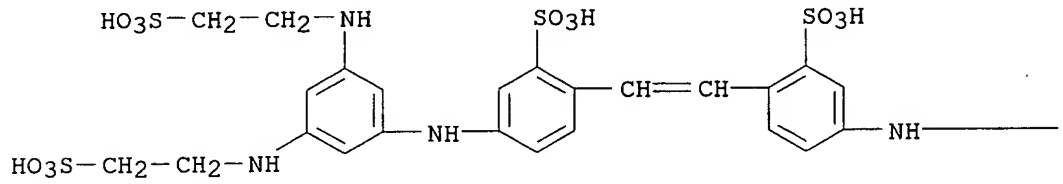


1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:70483

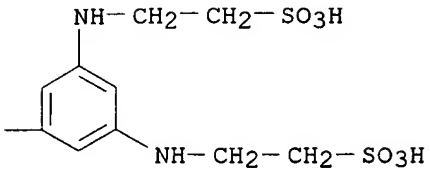
L3 ANSWER 27 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 439869-25-7 REGISTRY
CN Benzenesulfonic acid, 2,2'-(1,2-ethenediyl)bis[5-[[3,5-bis[(2-sulfoethyl)amino]phenyl]amino]-, hexasodium salt (9CI) (CA INDEX NAME)
MF C34 H42 N6 O18 S6 . 6 Na
SR CA
LC STN Files: CA, CAPLUS

PAGE 1-A



●6 Na

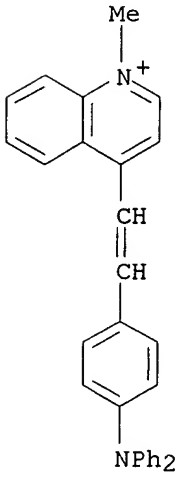
PAGE 1-B

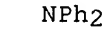


1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:70483

L3 ANSWER 28 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 439659-65-1 REGISTRY
CN Quinolinium, 4-[2-[4-(diphenylamino)phenyl]ethenyl]-1-methyl-, iodide (9CI) (CA INDEX NAME)
MF C30 H25 N2 . I
SR CA
LC STN Files: CA, CAPLUS





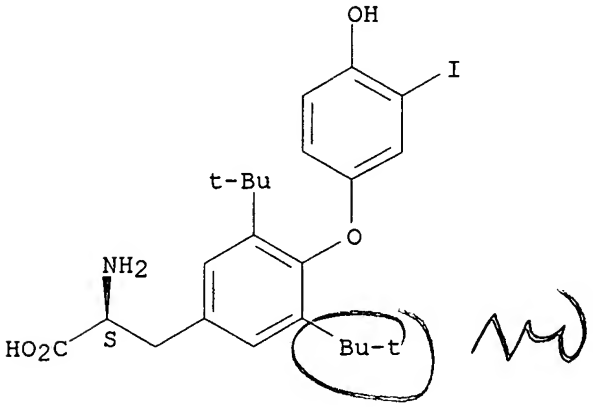
1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:64533

L3 ANSWER 29 OF 41052 REGISTRY COPYRIGHT 2002 ACS

RN 439590-31-5 REGISTRY
CN L-Tyrosine, 3,5-bis(1,1-dimethylethyl)-O-(4-hydroxy-3-iodophenyl)- (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C23 H30 I N O4
SR CA

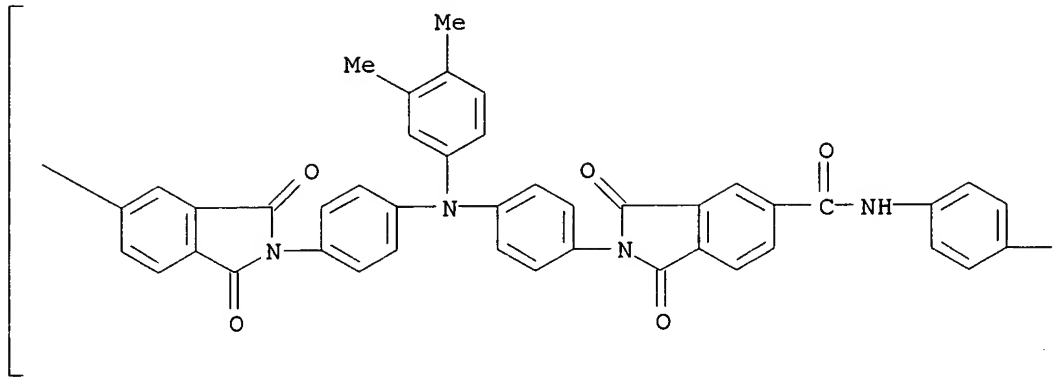
Absolute stereochemistry.



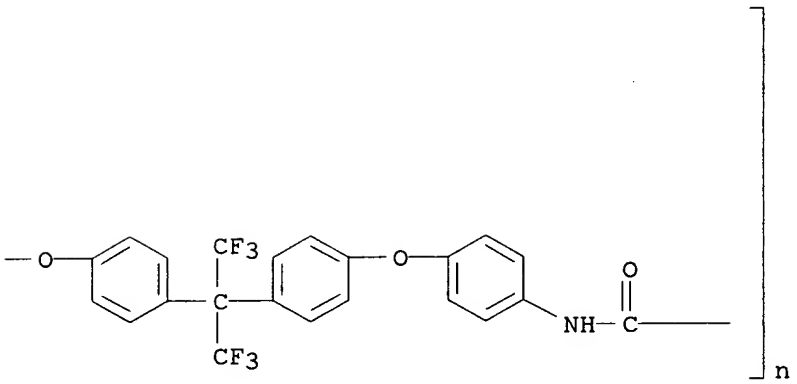

L3 ANSWER 30 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 439586-97-7 REGISTRY
CN Poly[(1,3-dihydro-1,3-dioxo-2H-isoindole-5,2-diyl)-1,4-phenylene[(3,4-dimethylphenyl)imino]-1,4-phenylene(1,3-dihydro-1,3-dioxo-2H-isoindole-2,5-diyl)carbonylimino-1,4-phenyleneoxy-1,4-phenylene[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene]-1,4-phenyleneoxy-1,4-phenyleneiminocarbonyl] (9CI) (CA INDEX NAME)
MF (C65 H41 F6 N5 O8)n
CI PMS
PCT Polyamide, Polyamine, Polyether, Polyimide
SR CA
LC STN Files: CA, CAPLUS

**RELATED POLYMERS AVAILABLE WITH POLYLINK**

PAGE 1-A



PAGE 1-B



1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:79317

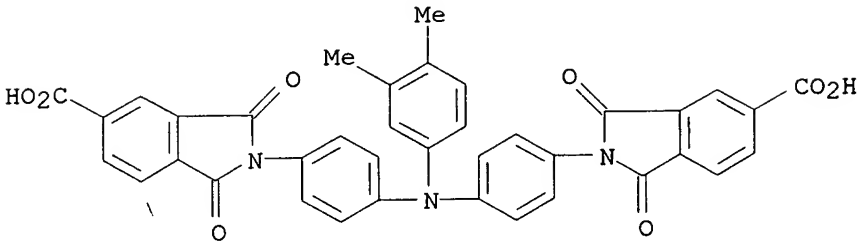
L3 ANSWER 31 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 439586-96-6 REGISTRY
CN 1H-Isoindole-5-carboxylic acid, 2,2'-[[(3,4-dimethylphenyl)imino]di-4,1-phenylene]bis[2,3-dihydro-1,3-dioxo-, polymer with 4,4'-[[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene]bis(4,1-phenyleneoxy)]bis[benzenamine] (9CI) (CA INDEX NAME)
MF (C38 H25 N3 O8 . C27 H20 F6 N2 O2)x
CI PMS
PCT Polyamide, Polyamide formed, Polyamine, Polyester, Polyester formed, Polyether, Polyimide
SR CA

LC STN Files: CA, CAPLUS

**RELATED POLYMERS AVAILABLE WITH POLYLINK**

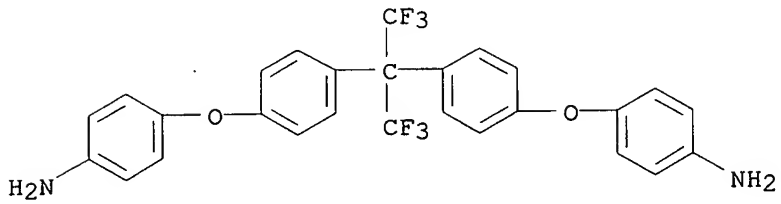
CM 1

CRN 439586-66-0
CMF C38 H25 N3 O8



CM 2

CRN 69563-88-8
CMF C27 H20 F6 N2 O2



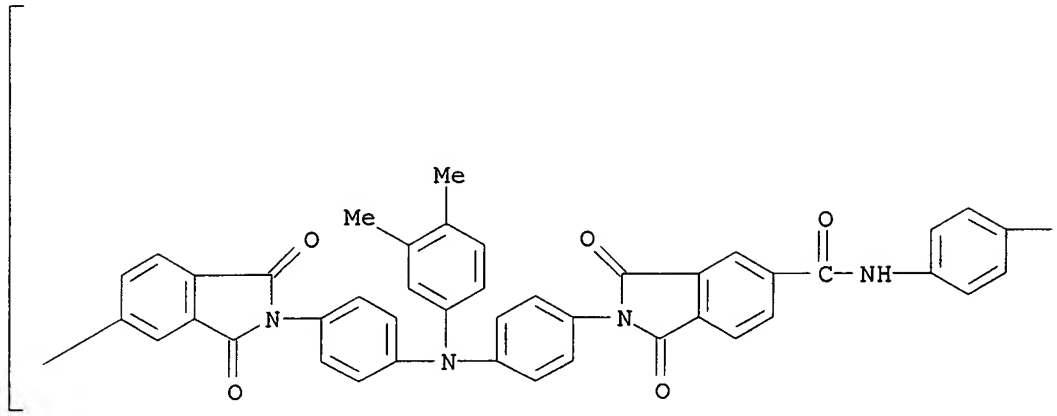
1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:79317

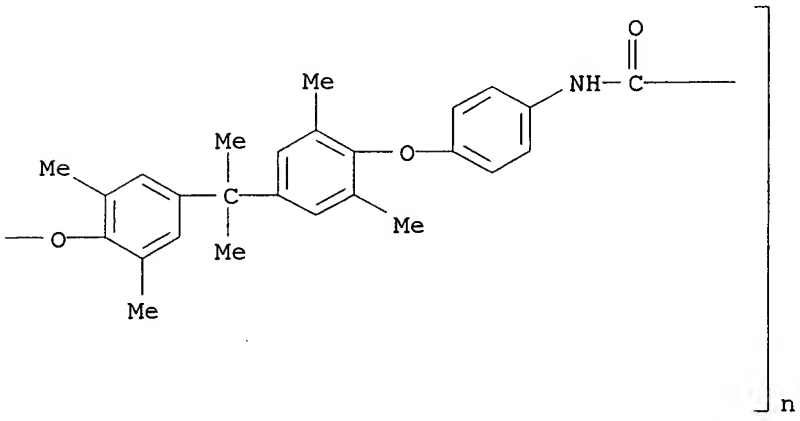
L3 ANSWER 32 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 439586-95-5 REGISTRY
CN Poly[(1,3-dihydro-1,3-dioxo-2H-isoindole-5,2-diyl)-1,4-phenylene[(3,4-dimethylphenyl)imino]-1,4-phenylene(1,3-dihydro-1,3-dioxo-2H-isoindole-2,5-diyl)carbonylimino-1,4-phenyleneoxy(2,6-dimethyl-1,4-phenylene)(1-methylethylidene)(3,5-dimethyl-1,4-phenylene)oxy-1,4-phenyleneiminocarbonyl] (9CI) (CA INDEX NAME)
MF (C69 H55 N5 O8)n
CI PMS
PCT Polyamide, Polyamine, Polyether, Polyimide
SR CA
LC STN Files: CA, CAPLUS

**RELATED POLYMERS AVAILABLE WITH POLYLINK**

PAGE 1-A



PAGE 1-B



1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:79317

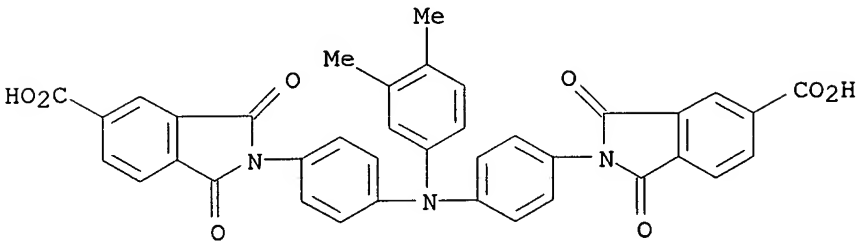
L3 ANSWER 33 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 439586-94-4 REGISTRY
CN 1H-Isoindole-5-carboxylic acid, 2,2'-[[(3,4-dimethylphenyl)imino]di-4,1-phenylene]bis[2,3-dihydro-1,3-dioxo-, polymer with 4,4'-[(1-methylethylidene)bis[(2,6-dimethyl-4,1-phenylene)oxy]]bis[benzenamine] (9CI) (CA INDEX NAME)

MF (C38 H25 N3 O8 . C31 H34 N2 O2)x
CI PMS
PCT Polyamide, Polyamide formed, Polyamine, Polyether, Polyimide
SR CA
LC STN Files: CA, CAPLUS

**RELATED POLYMERS AVAILABLE WITH POLYLINK**

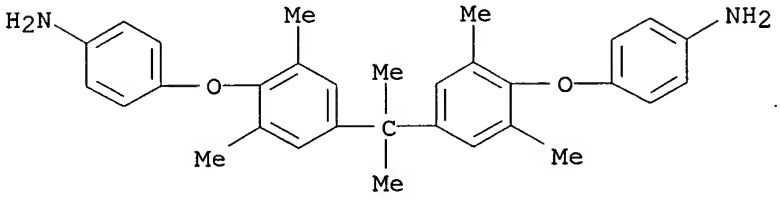
CM 1

CRN 439586-66-0
CMF C38 H25 N3 O8



CM 2

CRN 62488-02-2
CMF C31 H34 N2 O2



1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

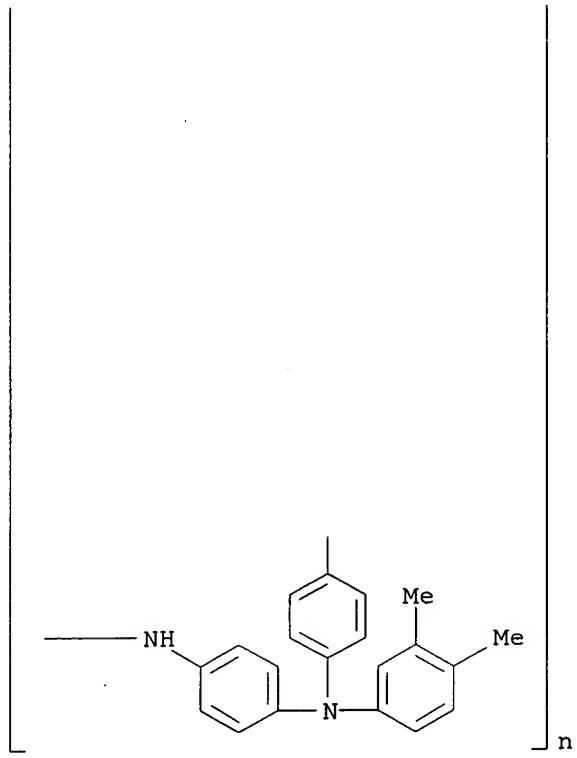
REFERENCE 1: 137:79317

L3 ANSWER 34 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 439586-78-4 REGISTRY
CN Poly[imino-1,4-phenylene[(3,4-dimethylphenyl)imino]-1,4-phenyleneiminocarbonyl-1,4-phenylene(1-methylethylidene)-1,4-phenylene] (9CI) (CA INDEX NAME)
MF (C36 H33 N3 O)n
CI PMS
PCT Polyamide, Polyamine

SR CA
LC STN Files: CA, CAPLUS

**RELATED POLYMERS AVAILABLE WITH POLYLINK**

* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY - AVAILABLE VIA OFFLINE PRINT *



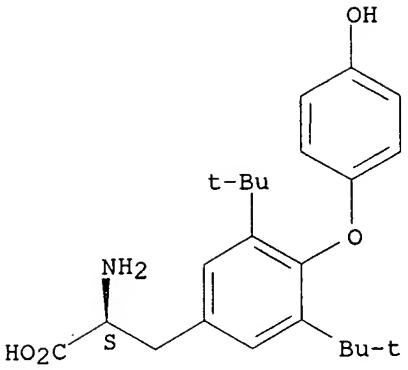

PAGE 2-A

1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:79317

L3 ANSWER 35 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 439573-89-4 REGISTRY
CN L-Tyrosine, 3,5-bis(1,1-dimethylethyl)-O-(4-hydroxyphenyl)- (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C23 H31 N O4
SR CA

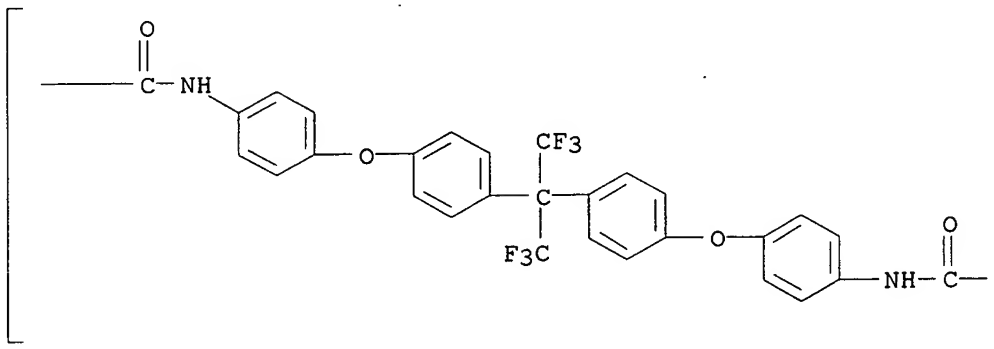
Absolute stereochemistry.



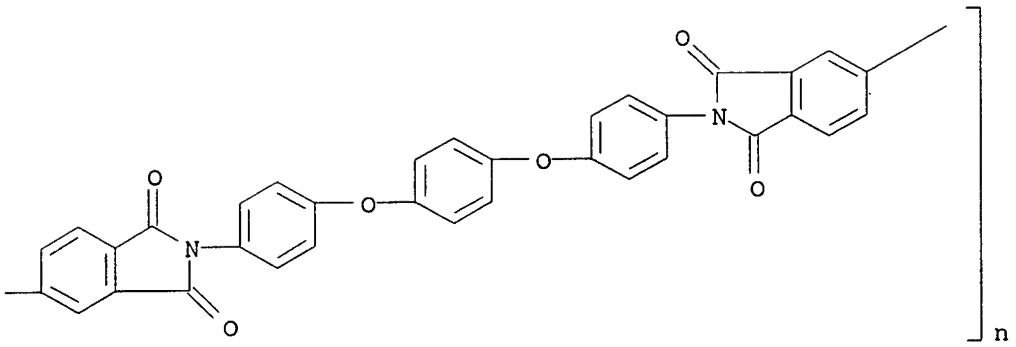

L3 ANSWER 36 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 439135-63-4 REGISTRY
CN Poly[(1,3-dihydro-1,3-dioxo-2H-isoindole-5,2-diyl)-1,4-phenyleneoxy-1,4-phenyleneoxy-1,4-phenylene(1,3-dihydro-1,3-dioxo-2H-isoindole-2,5-diyl)carbonylimino-1,4-phenyleneoxy-1,4-phenylene[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene]-1,4-phenyleneoxy-1,4-phenyleneiminocarbonyl] (9CI) (CA INDEX NAME)
MF (C63 H36 F6 N4 O10)n
CI PMS
PCT Polyamide, Polyether, Polyimide
SR CA
LC STN Files: CA, CAPLUS

**RELATED POLYMERS AVAILABLE WITH POLYLINK**

PAGE 1-A



PAGE 1-B



1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

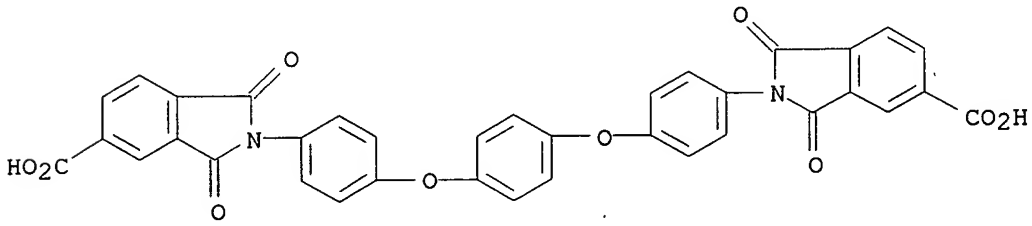
REFERENCE 1: 137:63554

L3 ANSWER 37 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 439135-62-3 REGISTRY
CN 1H-Isoindole-5-carboxylic acid, 2,2'-[1,4-phenylenebis(oxy-4,1-phenylene)]bis[2,3-dihydro-1,3-dioxo-, polymer with 4,4'-[[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene]bis(4,1-phenyleneoxy)]bis[benzenamine] (9CI) (CA INDEX NAME)
MF (C36 H20 N2 O10 . C27 H20 F6 N2 O2)x
CI PMS
PCT Polyamide, Polyamide formed, Polyester, Polyester formed, Polyether, Polyimide
SR CA
LC STN Files: CA, CAPLUS

**RELATED POLYMERS AVAILABLE WITH POLYLINK**

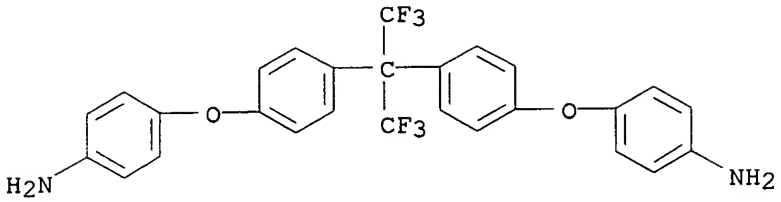
CM 1

CRN 130651-50-2
CMF C36 H20 N2 O10



CM 2

CRN 69563-88-8
CMF C27 H20 F6 N2 O2



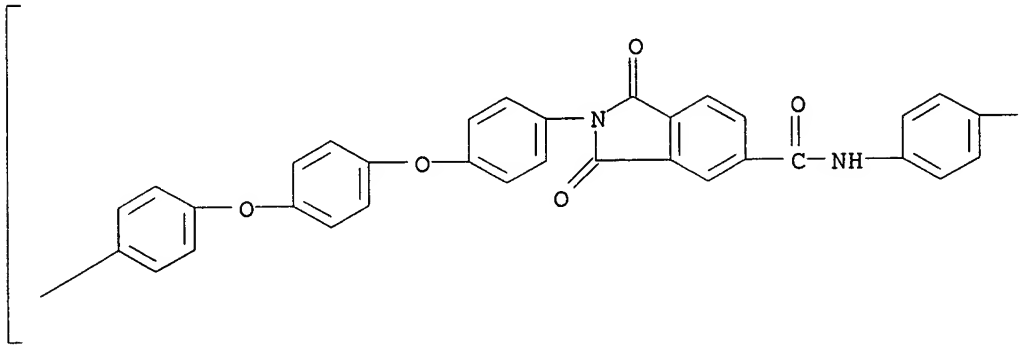
1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:63554

L3 ANSWER 38 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 439135-54-3 REGISTRY
CN Poly[(1,3-dihydro-1,3-dioxo-2H-isoindole-2,5-diyl)[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene](1,3-dihydro-1,3-dioxo-2H-isoindole-5,2-diyl)-1,4-phenyleneoxy-1,4-phenyleneoxy-1,4-phenylene(1,3-dihydro-1,3-dioxo-2H-isoindole-2,5-diyl)carbonylimino-1,4-phenyleneoxy-1,4-phenylene[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene]-1,4-phenyleneoxy-1,4-phenyleneiminocarbonyl(1,3-dihydro-1,3-dioxo-2H-isoindole-5,2-diyl)-1,4-phenyleneoxy-1,4-phenyleneoxy-1,4-phenylene] (9CI) (CA INDEX NAME)
MF (C100 H54 F12 N6 O16)n
CI PMS
PCT Polyamide, Polyether, Polyimide
SR CA
LC STN Files: CA, CAPLUS

**RELATED POLYMERS AVAILABLE WITH POLYLINK**

PAGE 1-A



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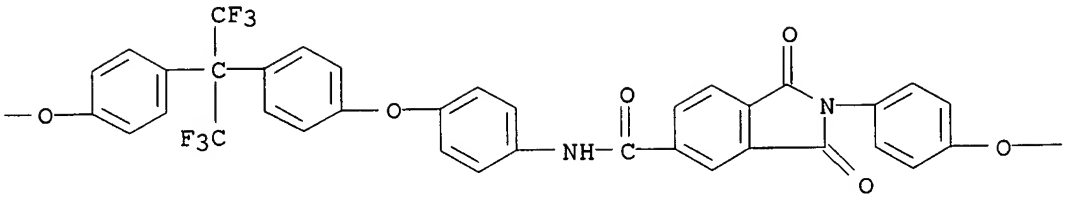

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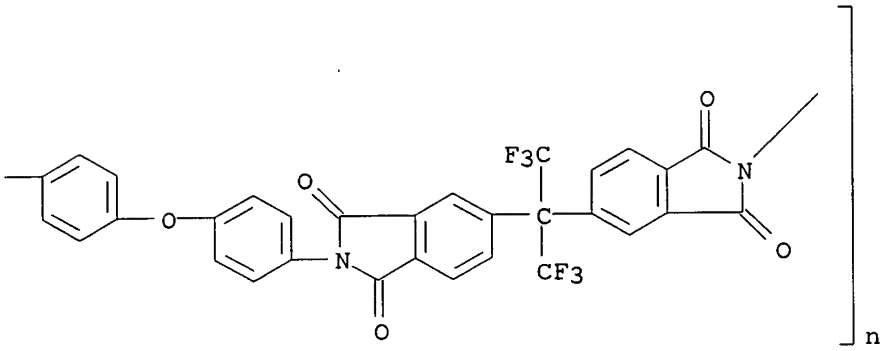


1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:63554

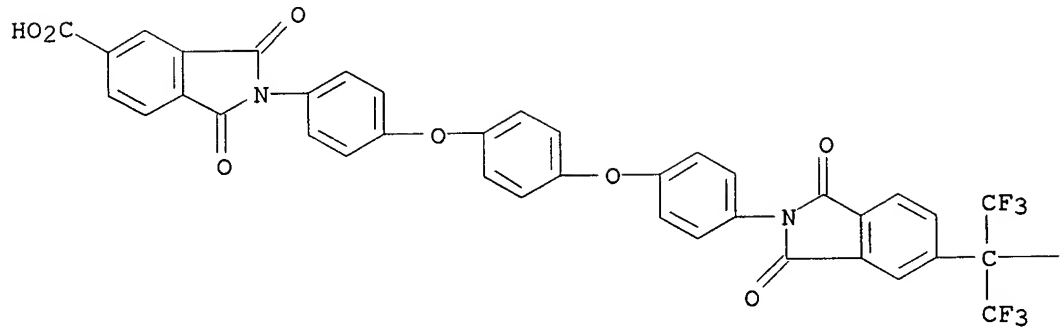
L3 ANSWER 39 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 439135-52-1 REGISTRY
CN 1H-Isoindole-5-carboxylic acid, 2,2'-[[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene]bis[(1,3-dihydro-1,3-dioxo-2H-isoindole-5,2-diyl)-4,1-phenyleneoxy-4,1-phenyleneoxy-4,1-phenylene]]bis[2,3-dihydro-1,3-dioxo-, polymer with 4,4'-[[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene]bis(4,1-phenyleneoxy)]bis[benzenamine] (9CI) (CA INDEX NAME)
MF (C73 H38 F6 N4 O16 . C27 H20 F6 N2 O2)x
CI PMS
PCT Polyamide, Polyamide formed, Polyester, Polyester formed, Polyether, Polyimide
SR CA
LC STN Files: CA, CAPLUS

**RELATED POLYMERS AVAILABLE WITH POLYLINK**

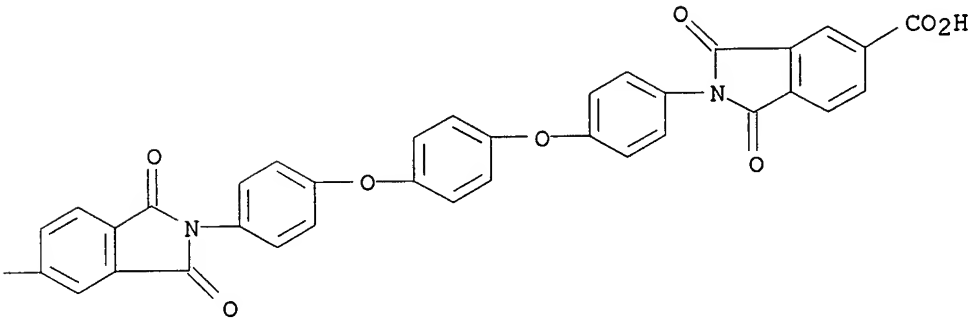
CM 1

CRN 439135-22-5
CMF C73 H38 F6 N4 O16

PAGE 1-A

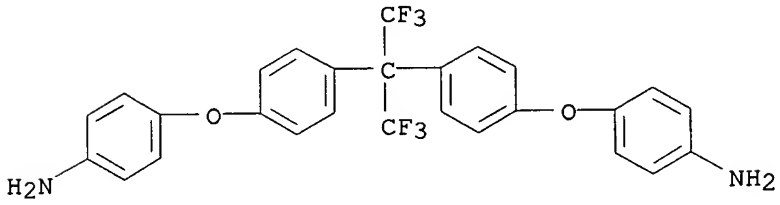


PAGE 1-B



CM 2

CRN 69563-88-8
CMF C27 H20 F6 N2 O2



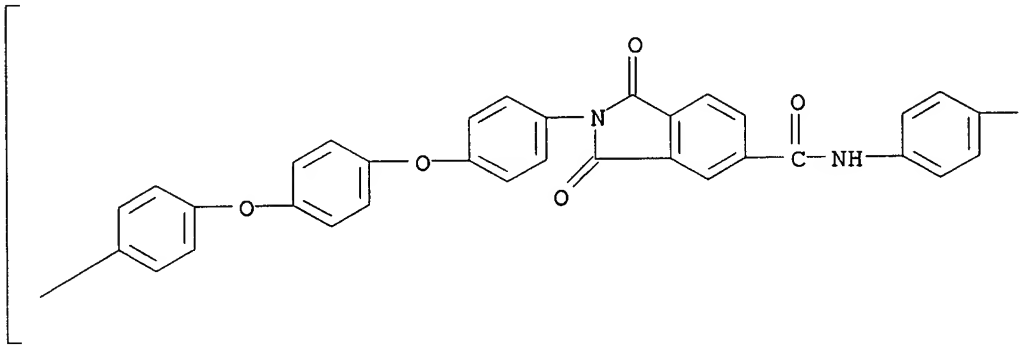
1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:63554

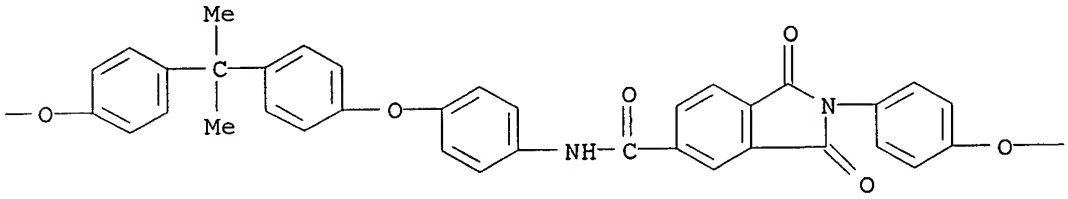
L3 ANSWER 40 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 439135-50-9 REGISTRY
CN Poly[(1,3-dihydro-1,3-dioxo-2H-isoindole-2,5-diyl)[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene](1,3-dihydro-1,3-dioxo-2H-isoindole-5,2-diyl)-1,4-phenyleneoxy-1,4-phenyleneoxy-1,4-phenylene(1,3-dihydro-1,3-dioxo-2H-isoindole-2,5-diyl)carbonylimino-1,4-phenyleneoxy-1,4-phenylene(1-methylethylidene)-1,4-phenyleneoxy-1,4-phenyleneiminocarbonyl(1,3-dihydro-1,3-dioxo-2H-isoindole-5,2-diyl)-1,4-phenyleneoxy-1,4-phenyleneoxy-1,4-phenylene] (9CI) (CA INDEX NAME)
MF (C100 H60 F6 N6 O16)n
CI PMS
PCT Polyamide, Polyether, Polyimide
SR CA
LC STN Files: CA, CAPLUS

**RELATED POLYMERS AVAILABLE WITH POLYLINK**

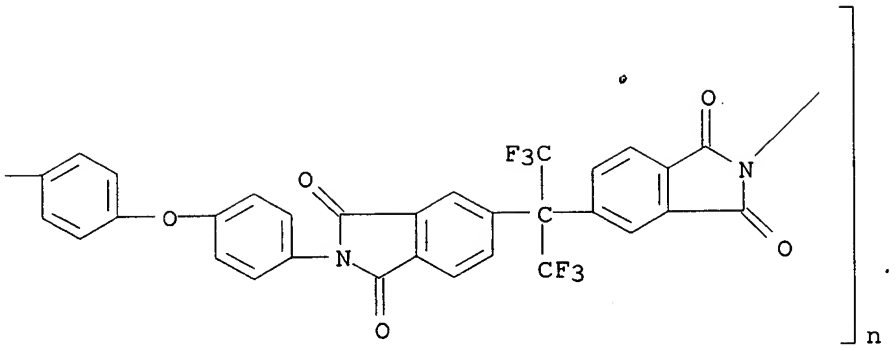
PAGE 1-A



PAGE 1-B



PAGE 1-C



1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:63554

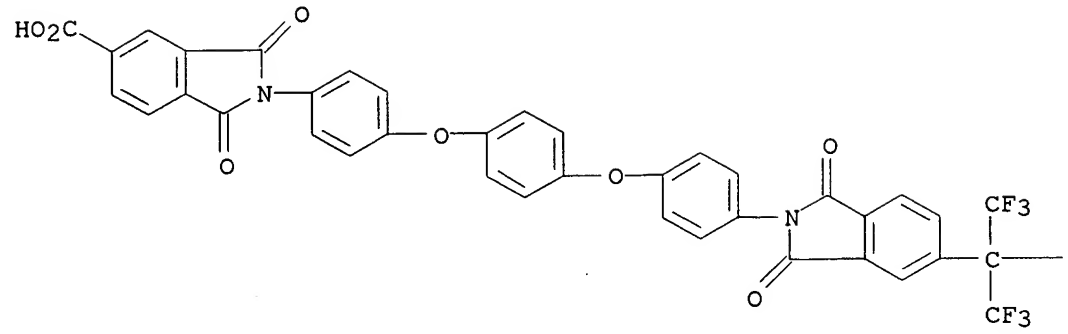
L3 ANSWER 41 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 439135-48-5 REGISTRY
CN 1H-Isoindole-5-carboxylic acid, 2,2'-[[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene]bis[(1,3-dihydro-1,3-dioxo-2H-isoindole-5,2-diyl)-4,1-phenyleneoxy-4,1-phenyleneoxy-4,1-phenylene]]bis[2,3-dihydro-1,3-dioxo-, polymer with 4,4'-[(1-methylethylidene)bis(4,1-phenyleneoxy)]bis[benzenamine] (9CI) (CA INDEX NAME)
MF (C73 H38 F6 N4 O16 . C27 H26 N2 O2)x
CI PMS
PCT Polyamide, Polyamide formed, Polyester, Polyester formed, Polyether, Polyimide
SR CA
LC STN Files: CA, CAPLUS

**RELATED POLYMERS AVAILABLE WITH POLYLINK**

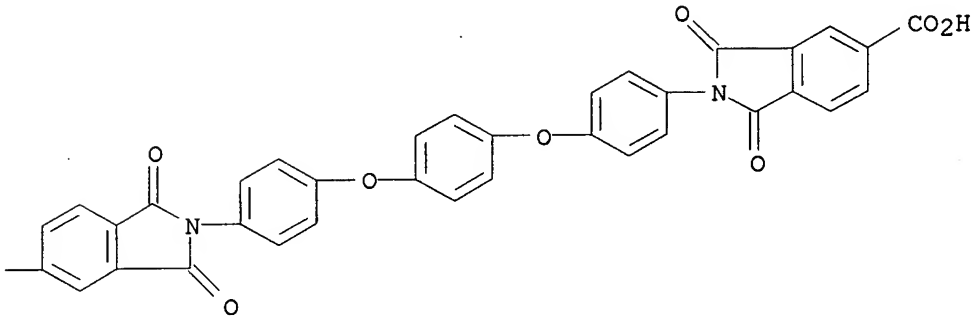
CM 1

CRN 439135-22-5
CMF C73 H38 F6 N4 O16

PAGE 1-A

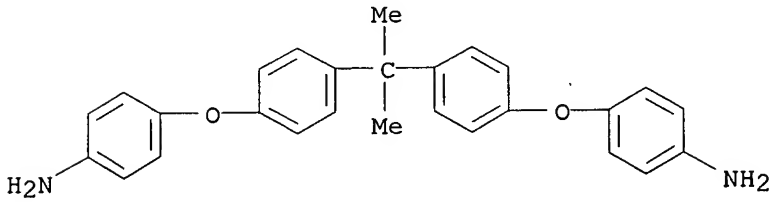


PAGE 1-B



CM 2

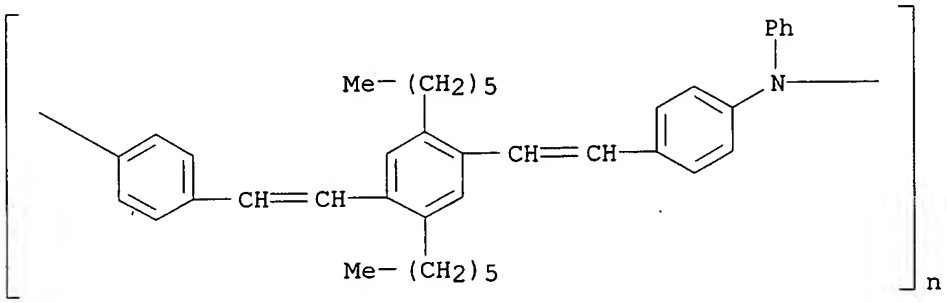
CRN 13080-86-9
CMF C27 H26 N2 O2



1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:63554

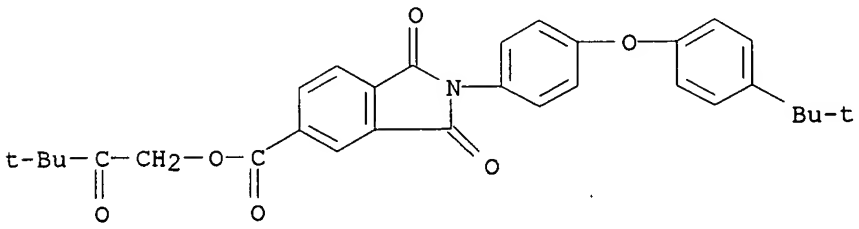
L3 ANSWER 42 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 439101-62-9 REGISTRY
CN Poly[(phenylimino)-1,4-phenylene-1,2-ethenediyl(2,5-dihexyl-1,4-phenylene)-1,2-ethenediyl-1,4-phenylene] (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 4,4'-Diformyltriphenylamine-2,5-dihexyl-1,4-xylylenebis(diethylphosphonate) copolymer, SRU
MF (C40 H45 N)n
CI PMS
PCT Polyamine
SR CA
LC STN Files: CA, CAPLUS




1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:63566

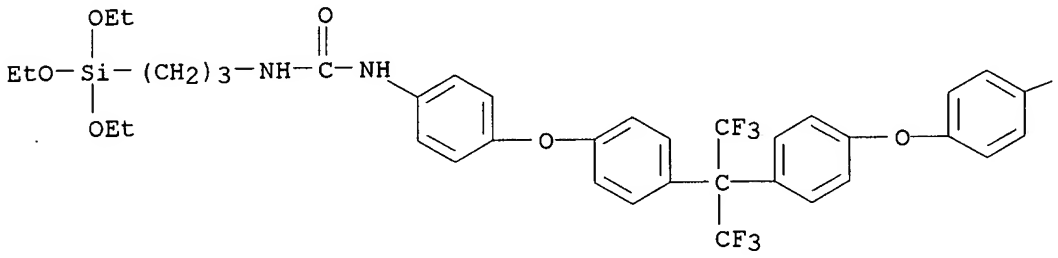
L3 ANSWER 43 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 439092-28-1 REGISTRY
CN 1H-Isoindole-5-carboxylic acid, 2-[4-[4-(1,1-dimethylethyl)phenoxy]phenyl]-2,3-dihydro-1,3-dioxo-, 3,3-dimethyl-2-oxobutyl ester (9CI) (CA INDEX NAME)
FS 3D CONCORD
MF C31 H31 N O6
SR Chemical Library
LC STN Files: CHEMCATS



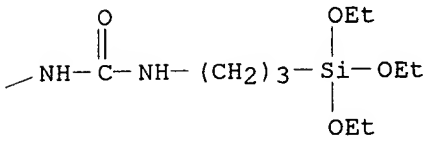
**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

L3 ANSWER 44 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 438633-77-3 REGISTRY
CN Urea, N,N''-[[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene]bis(4,1-phenyleneoxy-4,1-phenylene)]bis[N'-[3-(triethoxysilyl)propyl]- (9CI) (CA INDEX NAME)
FS 3D CONCORD
MF C47 H62 F6 N4 O10 Si2
SR CA
LC STN Files: CA, CAPLUS

PAGE 1-A



PAGE 1-B



**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

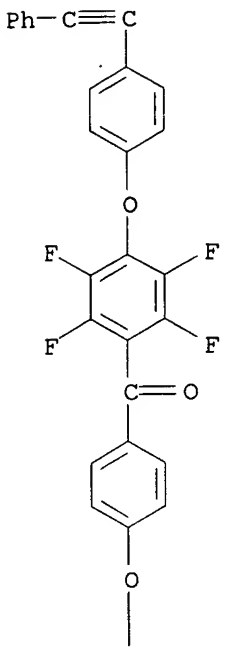
1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:47971

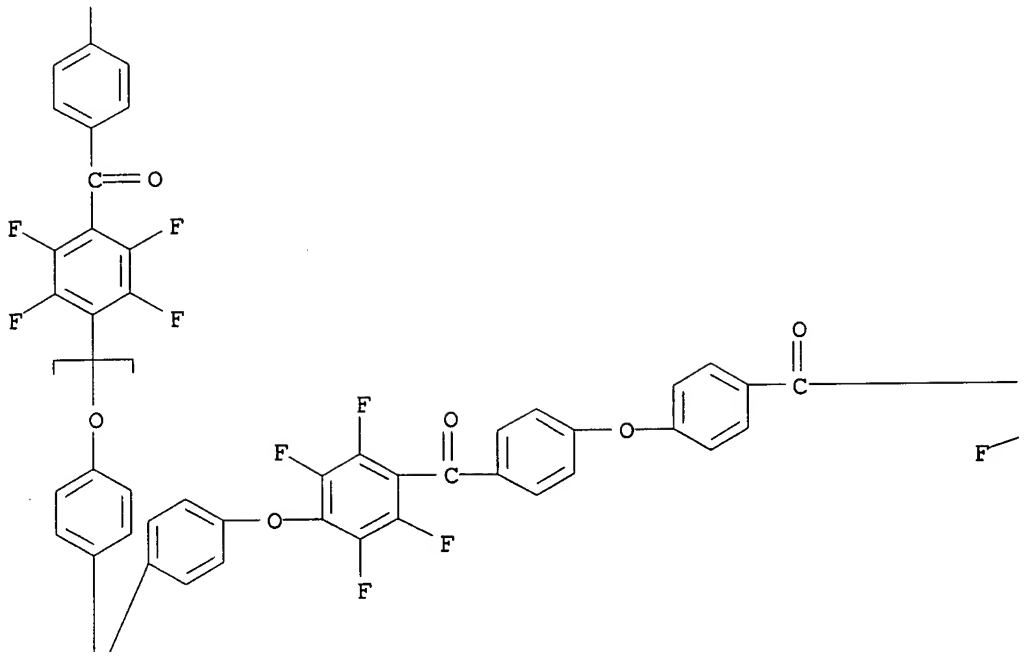
L3 ANSWER 45 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 438588-47-7 REGISTRY
CN Poly[oxy-1,4-phenylene-9H-fluoren-9-ylidene-1,4-phenyleneoxy(2,3,5,6-tetrafluoro-1,4-phenylene)carbonyl-1,4-phenyleneoxy-1,4-phenylenecarbonyl(2,3,5,6-tetrafluoro-1,4-phenylene)], .alpha.-[2,3,5,6-tetrafluoro-4-[4-[4-[2,3,5,6-tetrafluoro-4-[4-(phenylethynyl)phenoxy]benzoyl]phenoxy]benzoyl]phenyl]-.omega.-[4-(phenylethynyl)phenoxy]- (9CI) (CA INDEX NAME)
MF (C51 H24 F8 O5)n C54 H26 F8 O5

CI PMS
PCT Polyether, Polyketone
SR CA
LC STN Files: CA, CAPLUS

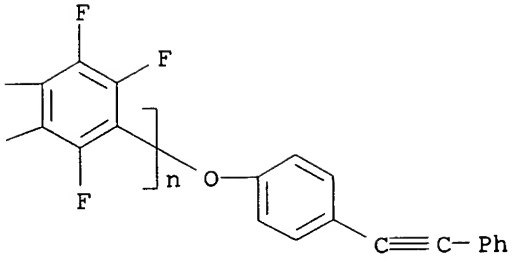
PAGE 1-A



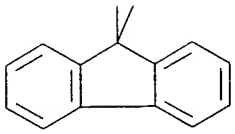
PAGE 2-A



PAGE 2-B



PAGE 3-A

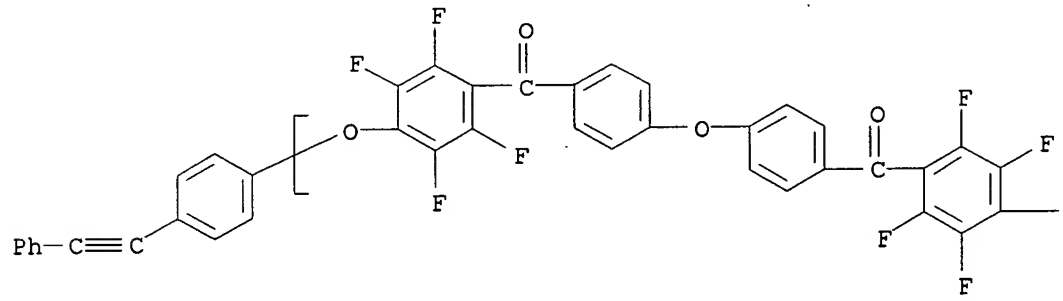


1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:47543

L3 ANSWER 46 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 438588-46-6 REGISTRY
CN Poly[oxy(2,3,5,6-tetrafluoro-1,4-phenylene)carbonyl-1,4-phenyleneoxy-1,4-phenylenecarbonyl(2,3,5,6-tetrafluoro-1,4-phenylene)oxy-1,4-phenylene[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene]-1,4-phenylene], .alpha.-[4-(phenylethynyl)phenyl]-.omega.-[2,3,5,6-tetrafluoro-4-[4-[4-[2,3,5,6-tetrafluoro-4-[4-(phenylethynyl)phenoxy]benzoyl]phenoxy]benzoyl]phenoxy]- (9CI) (CA INDEX NAME)
MF (C41 H16 F14 O5)n C54 H26 F8 O5
CI PMS
PCT Polyether, Polyketone
SR CA
LC STN Files: CA, CAPLUS

PAGE 1-A



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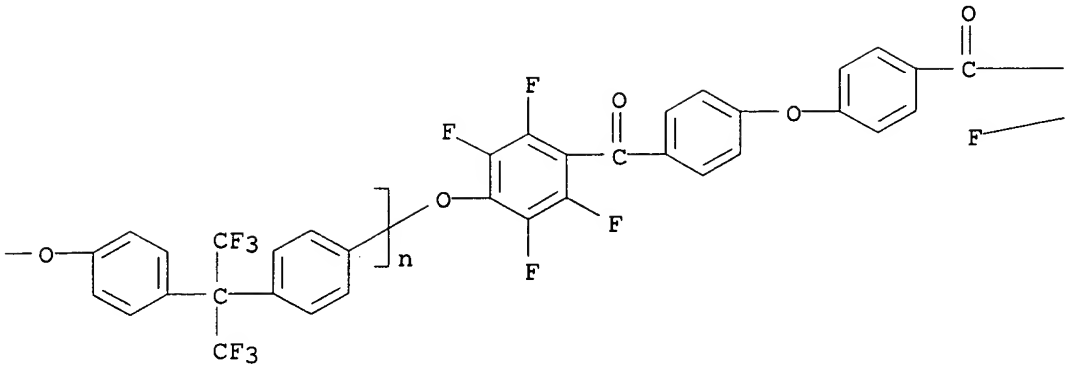
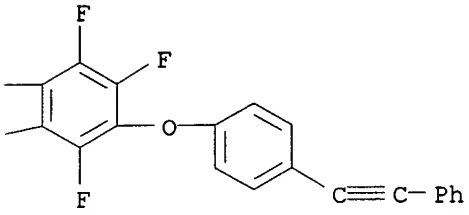


PAGE 1-C

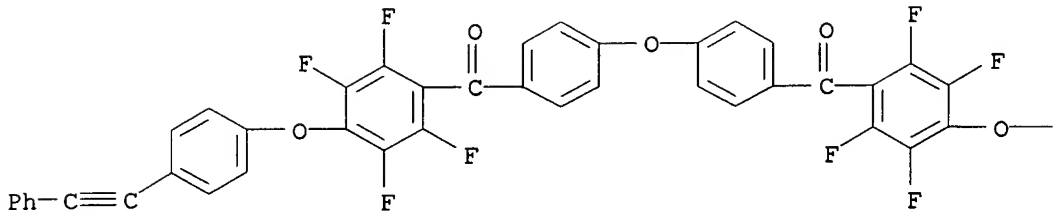


1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:47543

L3 ANSWER 47 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 438588-45-5 REGISTRY
CN Methanone, (oxydi-4,1-phenylene)bis[[2,3,5,6-tetrafluoro-4-[4-(phenylethynyl)phenoxy]phenyl]- (9CI) (CA INDEX NAME)
FS 3D CONCORD
MF C54 H26 F8 O5
SR CA
LC STN Files: CA, CAPLUS

PAGE 1-A



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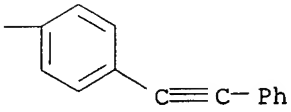


**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

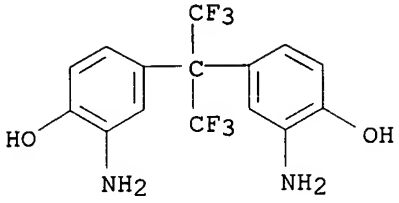
1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:47543

L3 ANSWER 48 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 438572-95-3 REGISTRY
CN Benzoic acid, 3,5-diamino-, polymer with 5,5'-carbonylbis[1,3-isobenzofurandione], 4,4'-[(1-methylethylidene)bis(4,1-phenyleneoxy)]bis[benzenamine], 2,4,8,10-tetraoxaspiro[5.5]undecane-3,9-dipropanamine and 4,4'-[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene]bis[2-aminophenol] (9CI) (CA INDEX NAME)
MF (C27 H26 N2 O2 . C17 H6 O7 . C15 H12 F6 N2 O2 . C13 H26 N2 O4 . C7 H8 N2 O2)x
CI PMS
PCT Polyamic acid, Polyamic acid formed, Polyamide, Polyamide formed, Polyester, Polyester formed, Polyether, Polyimide, Polyimide formed, Polyketone
SR CA
LC STN Files: CA, CAPLUS

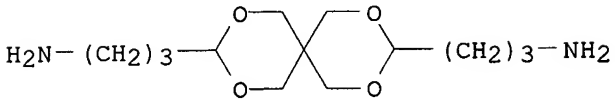
CM 1

CRN 83558-87-6
CMF C15 H12 F6 N2 O2



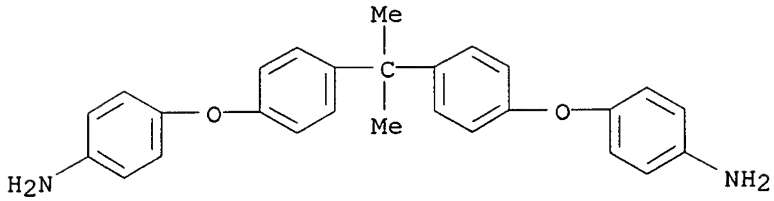
CM 2

CRN 21587-74-6
CMF C13 H26 N2 O4



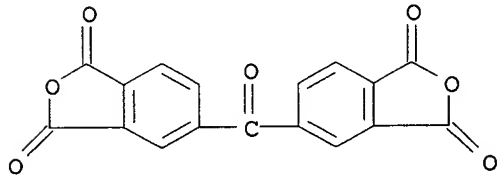
CM 3

CRN 13080-86-9
CMF C27 H26 N2 O2



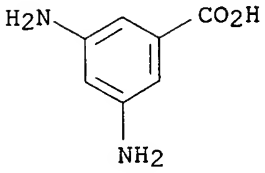
CM 4

CRN 2421-28-5
CMF C17 H6 O7



CM 5

CRN 535-87-5
CMF C7 H8 N2 O2

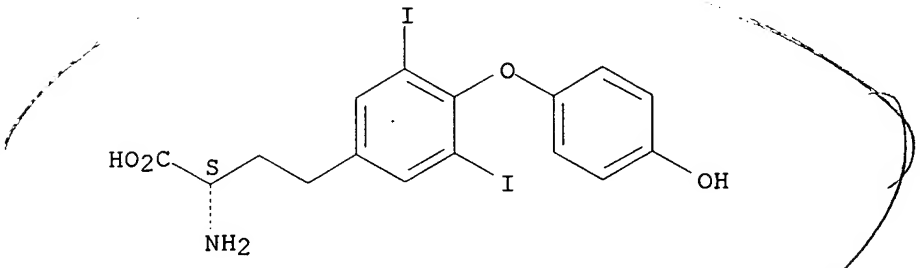


1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:48660

L3 ANSWER 49 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 438552-43-3 REGISTRY
CN Benzenebutanoic acid, .alpha.-amino-4-(4-hydroxyphenoxy)-3,5-diiodo-, (.alpha.S)- (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C16 H15 I2 N O4
SR CA

Absolute stereochemistry.



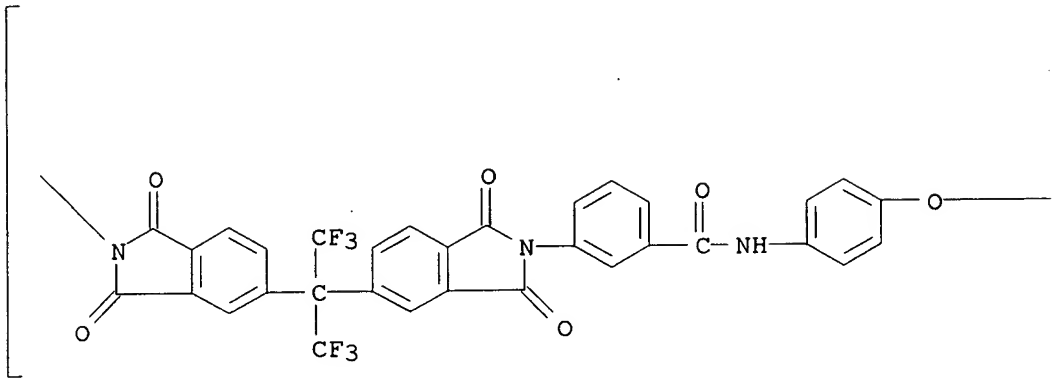
**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

L3 ANSWER 50 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 438547-87-6 REGISTRY
CN Poly[(1,3-dihydro-1,3-dioxo-2H-isoindole-2,5-diyl)[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene](1,3-dihydro-1,3-dioxo-2H-isoindole-5,2-diyl)-1,3-phenylenecarbonylimino-1,4-phenyleneoxy-1,4-phenylene[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene]-1,4-phenyleneoxy-1,4-phenyleneiminocarbonyl-1,3-phenylene] (9CI) (CA INDEX NAME)
MF (C60 H32 F12 N4 O8)n
CI PMS
PCT Polyamide, Polyether, Polyimide

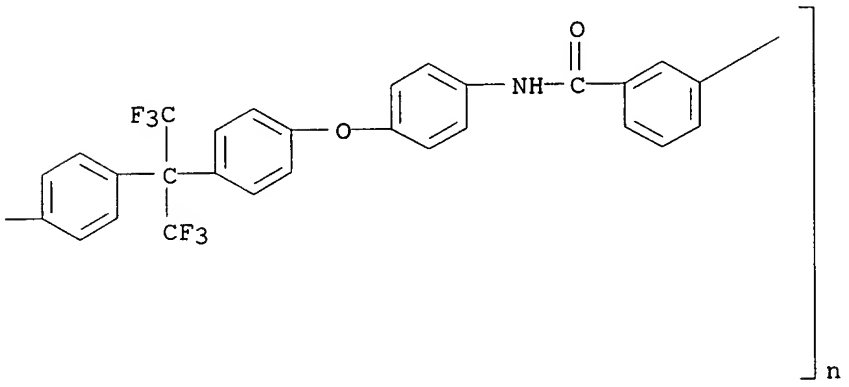
SR CA
LC STN Files: CA, CAPLUS

**RELATED POLYMERS AVAILABLE WITH POLYLINK**

PAGE 1-A



PAGE 1-B



1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:47539

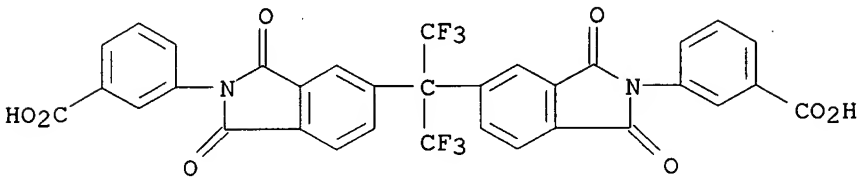
L3 ANSWER 51 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 438547-86-5 REGISTRY
CN Benzoic acid, 3,3'-[[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene]bis(1,3-dihydro-1,3-dioxo-2H-isoindole-5,2-diyl)]bis-, polymer with 4,4'-[[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene]bis(4,1-phenyleneoxy)]bis[benzenamine] (9CI) (CA INDEX NAME)

MF (C33 H16 F6 N2 O8 . C27 H20 F6 N2 O2)x
CI PMS
PCT Polyamide, Polyamide formed, Polyester, Polyester formed, Polyether, Polyimide
SR CA
LC STN Files: CA, CAPLUS

**RELATED POLYMERS AVAILABLE WITH POLYLINK**

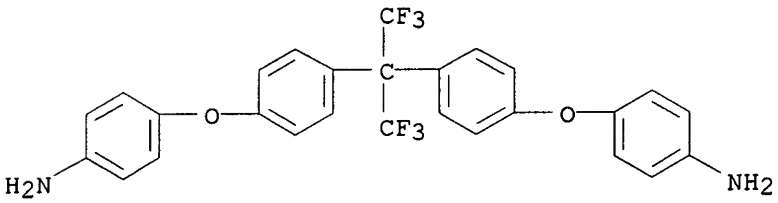
CM 1

CRN 159523-62-3
CMF C33 H16 F6 N2 O8



CM 2

CRN 69563-88-8
CMF C27 H20 F6 N2 O2



1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:47539

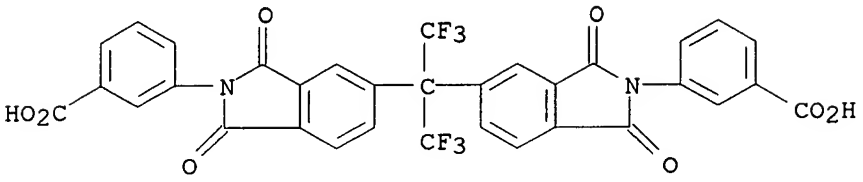
L3 ANSWER 52 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 438547-85-4 REGISTRY
CN Benzoic acid, 3,3'-[[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene]bis(1,3-dihydro-1,3-dioxo-2H-isoindole-5,2-diyl)]bis-, polymer with 4,4'-[(1-methylethylidene)bis(4,1-phenyleneoxy)]bis[benzenamine] (9CI) (CA INDEX NAME)
MF (C33 H16 F6 N2 O8 . C27 H26 N2 O2)x
CI PMS
PCT Polyamide, Polyamide formed, Polyester, Polyester formed, Polyether, Polyimide

SR CA
LC STN Files: CA, CAPLUS

**RELATED POLYMERS AVAILABLE WITH POLYLINK**

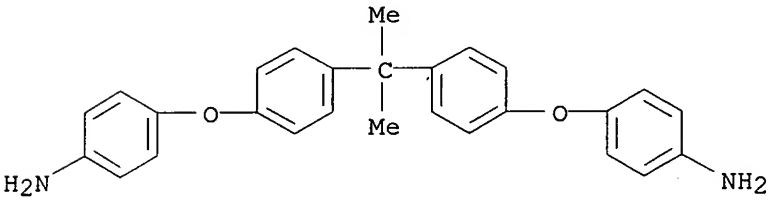
CM 1

CRN 159523-62-3
CMF C33 H16 F6 N2 O8



CM 2

CRN 13080-86-9
CMF C27 H26 N2 O2



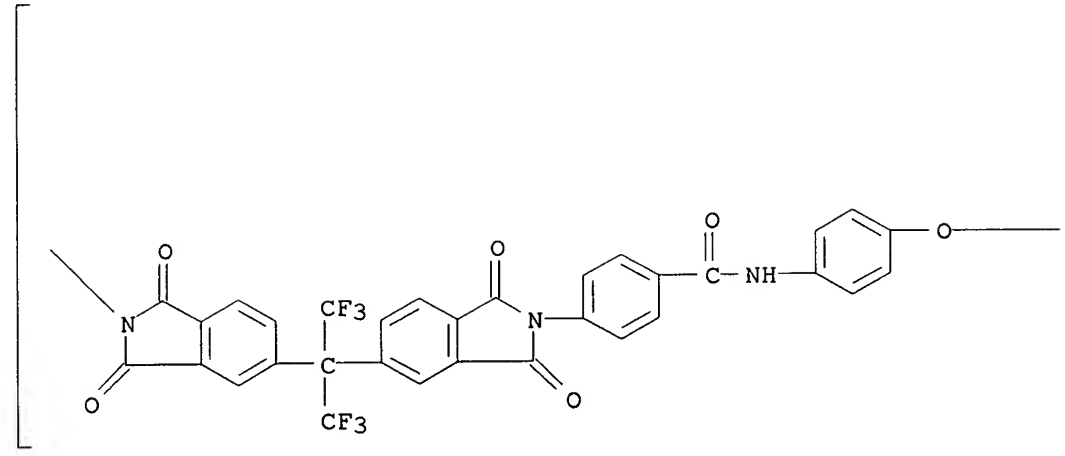
1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:47539

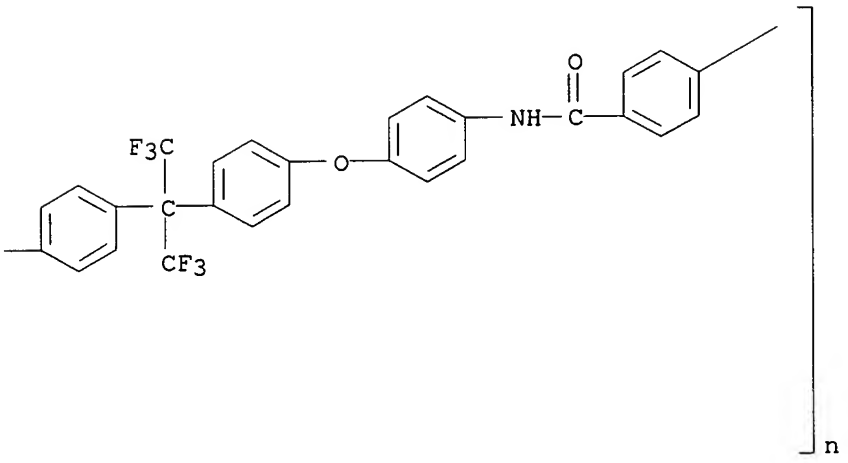
L3 ANSWER 53 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 438547-74-1 REGISTRY
CN Poly[(1,3-dihydro-1,3-dioxo-2H-isoindole-2,5-diyl)[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene](1,3-dihydro-1,3-dioxo-2H-isoindole-5,2-diyl)-1,4-phenylenecarbonylimino-1,4-phenyleneoxy-1,4-phenylene[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene]-1,4-phenyleneoxy-1,4-phenyleneiminocarbonyl-1,4-phenylene] (9CI) (CA INDEX NAME)
MF (C60 H32 F12 N4 O8)n
CI PMS
PCT Polyamide, Polyether, Polyimide
SR CA
LC STN Files: CA, CAPLUS

**RELATED POLYMERS AVAILABLE WITH POLYLINK**

PAGE 1-A



PAGE 1-B



1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:47539

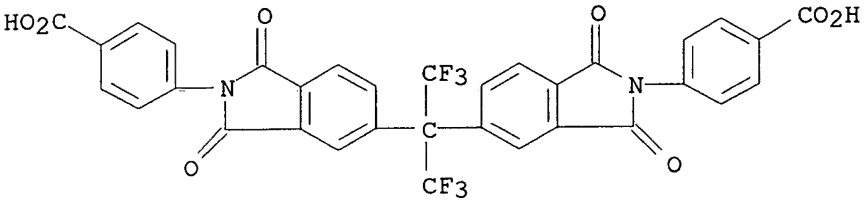
L3 ANSWER 54 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 438547-73-0 REGISTRY
CN Benzoic acid, 4,4'-[[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene]bis(1,3-dihydro-1,3-dioxo-2H-isoindole-5,2-diyl)]bis-, polymer with

4,4'-[[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene]bis(4,1-phenyleneoxy)]bis[benzenamine] (9CI) (CA INDEX NAME)
MF (C33 H16 F6 N2 O8 . C27 H20 F6 N2 O2)x
CI PMS
PCT Polyamide, Polyamide formed, Polyester, Polyester formed, Polyether, Polyimide
SR CA
LC STN Files: CA, CAPLUS

**RELATED POLYMERS AVAILABLE WITH POLYLINK**

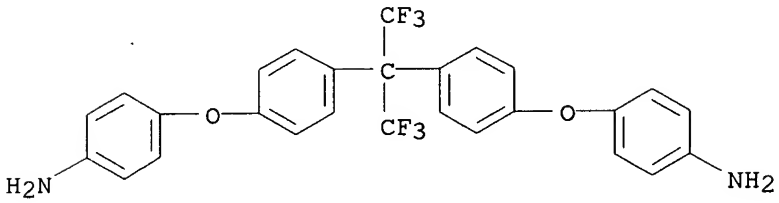
CM 1

CRN 133532-50-0
CMF C33 H16 F6 N2 O8



CM 2

CRN 69563-88-8
CMF C27 H20 F6 N2 O2



1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:47539

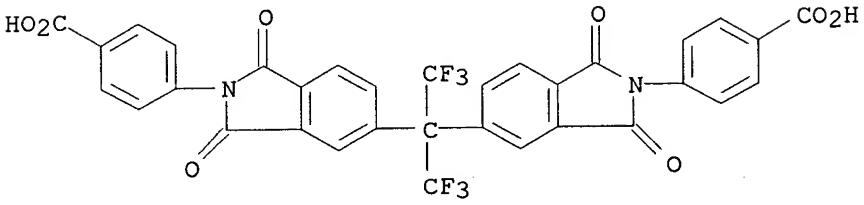
L3 ANSWER 55 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 438547-72-9 REGISTRY
CN Benzoic acid, 4,4'-[[2,2,2-trifluoro-1-(trifluoromethyl)ethylidene]bis(1,3-dihydro-1,3-dioxo-2H-isoindole-5,2-diyl)]bis-, polymer with 4,4'-[(1-methylethylidene)bis(4,1-phenyleneoxy)]bis[benzenamine] (9CI) (CA INDEX NAME)
MF (C33 H16 F6 N2 O8 . C27 H26 N2 O2)x

CI PMS
PCT Polyamide, Polyamide formed, Polyester, Polyester formed, Polyether, Polyimide
SR CA
LC STN Files: CA, CAPLUS

**RELATED POLYMERS AVAILABLE WITH POLYLINK**

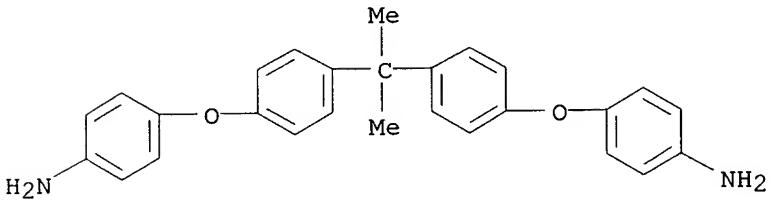
CM 1

CRN 133532-50-0
CMF C33 H16 F6 N2 O8



CM 2

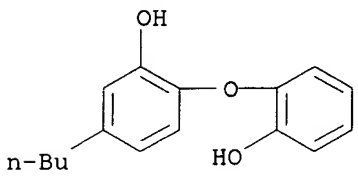
CRN 13080-86-9
CMF C27 H26 N2 O2



1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:47539

L3 ANSWER 56 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 438543-90-9 REGISTRY
CN Phenol, 5-butyl-2-(2-hydroxyphenoxy)- (9CI) (CA INDEX NAME)
MF C16 H18 O3
SR CA
LC STN Files: CA, CAPLUS

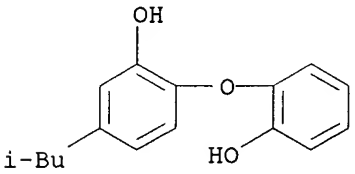



**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:51982

L3 ANSWER 57 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 438543-88-5 REGISTRY
CN Phenol, 2-(2-hydroxyphenoxy)-5-(2-methylpropyl)- (9CI) (CA INDEX NAME)
MF C16 H18 O3
SR CA
LC STN Files: CA, CAPLUS

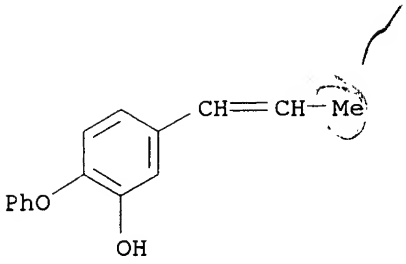



**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:51982

L3 ANSWER 58 OF 41052 REGISTRY COPYRIGHT 2002 ACS
RN 438543-86-3 REGISTRY
CN Phenol, 2-phenoxy-5-(1-propenyl)- (9CI) (CA INDEX NAME)
FS 3D CONCORD
MF C15 H14 O2
SR CA
LC STN Files: CA, CAPLUS




**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 137:51982